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NEWS 11	Jun 10	PCTFULL has been reloaded
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L1          202 TAB1 (10A) TAK1 (10A) (BINDING OR BIND)

=> s  tab1 (5A) tak1
  47 FILES SEARCHED...
L2          270 TAB1 (5A) TAK1

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L4            3 L3 AND REVIEW

=> d l4 1-3 bib ab

L4   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2002 ACS
AN   1999:712337  CAPLUS
DN   131:317866
TI   Functional role for TAB1-TAK1 in TGF-.beta. signaling
```

AU Shibuya, Hiroshi
CS Div. Morphogenesis, Natl. Inst. Basic Biol., Nishigonaka 38, Myodaiji, Okazaki, 444-8585, Japan
SO Seikagaku (1999), 71(10), 1205-1212
CODEN: SEIKAQ; ISSN: 0037-1017
PB Nippon Seikagakkai
DT Journal; General Review
LA Japanese
AB A **review** with 13 refs., on roles of TAB1-TAK1, involved in the TGF-.beta./BMP signaling pathway, in the Xenopus embryogenesis, discussing functions of TAB1-TAK1 in TGF-.beta. signaling pathway, roles of TAB1 and TAK1 in the early development, and regulation of TAB1-TAK1-mediated apoptotic signals by XIAP, a member of inhibitor of apoptosis protein family.

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS
AN 1996:715668 CAPLUS
DN 126:43981
TI MAP kinase kinase kinase, TAK1 functions in TGF-.beta.-mediated signal transduction
AU Irie, Kenji; Shibuya, Hiroshi
CS Fac. Sci., Nagoya Univ., Nagoya, 464-01, Japan
SO Jikken Igaku (1996), 14(19), 2616-2622
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; General Review
LA Japanese
AB A **review** with 21 refs., on isolation and function of a new TAK1 kinase and its activator, **TAK1-binding** protein 1 (**TAB1**), in TGF-.beta.-mediated signal transduction pathway. Involvement of TAK1 and TAB1 in TGF-.beta.-stimulated PAI-1 gene expression is also discussed.

L4 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS
AN 1996:530982 CAPLUS
DN 125:212786
TI New MAPKKK, TAK1, functions in TGF-.beta. signal transduction
AU Yamaguchi, Kyoko; Shirakabe, Kyoko
CS Sch. Sci., Nagoya Univ., Nagoya, 464-01, Japan
SO Jikken Igaku (1996), 14(13), 1846-1851
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; General Review
LA Japanese
AB A **review**, with 9 refs., on search for mammalian novel MAPKKK (mitogen-activated protein kinase kinase kinase) by using MAPK (MAP kinase) cascade of yeast and isolation of TAK1 (TGF-.beta. activated kinase 1) as an activating factor for Ste7-P368, effect of TAK1 on expression of PAI-1 gene, activation of TAK1 by stimulation with TGF-.beta., MAPK cascade by TGF-.beta. stimulation, identification of **TAB1** (**TAK1-binding** protein 1), and role of TAB1 in TGF-.beta. signal transduction.

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     1
AN   2002:583022  BIOSIS
DN   PREV200200583022
TI   Method of screening TGF-beta inhibitory substances.
AU   Ono, Koichiro (1); Ohtomo, Toshihiko; Tsuchiya, Masayuki
CS   (1) Gotenba Japan
     ASSIGNEE: Chugai Seiyaku Kabushiki Kaisha, Tokyo, Japan
PI   US 6451617 September 17, 2002
SO   Official Gazette of the United States Patent and Trademark Office Patents,
     (Sep. 17, 2002) Vol. 1262, No. 3, pp. No Pagination.
     http://www.uspto.gov/web/menu/patdata.html. e-file.
     ISSN: 0098-1133.
DT   Patent
LA   English
AB   A method for screening substances that inhibit binding
     between a TAK1 polypeptide and a TAB1 polypeptide,
     which comprises contacting the TAB1 polypeptide to the TAK1 polypeptide
     and a test sample and then detecting or determining the TAK1 polypeptide
     that is bound to the TAB1 polypeptide.
```

L6 ANSWER 2 OF 66 IFIPAT COPYRIGHT 2002 IFI DUPLICATE 2
AN 10211917 IFIPAT;IFIUDB;IFICDB
TI METHOD OF SCREENING TGF-BETA-INHIBITING SUBSTANCES
INF Ohtomo; Toshihiko, Gotenba-shi, JP
Ono; Koichiro, Gotenba-shi, JP
Tsuchiya; Masayuki, Gotenba-shi, JP
IN Ohtomo Toshihiko (JP); Ono Koichiro (JP); Tsuchiya Masayuki (JP)
PAF CHUGAI SEIYAKU KABUSHIKI KAISHA
PA Chugai Seiyaku K K JP (17384)
AG FOLEY AND LARDNER SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007, US
PI US 2002155624 A1 20021024
AI US 2002-158895 20020603
RLI US 2000-529279 20000411 CONTINUATION PENDING
WO 1998-JP4796 19981022 Section 371 PCT Filing UNKNOWN
PRAI JP 1997-290188 19971022
FI US 2002155624 20021024
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
CLMN 36
GI 14 Figure(s).

FIG. 1 is a diagram showing the construction of human TAB1-FLAG and human TAK1-6 x His.

FIG. 2 is a graph showing binding between human TAK1-FLAG and human MBP-TAB1C-FLAG.

FIG. 3 is a graph showing binding between human TAB1-FLAG and human TAK1-6 x His.

FIG. 4 is a graph showing the activity of inhibition of binding between human TAK1-6 x His and human MBP-TAB1C-FLAG, determined using TAB1-FLAG as an inhibiting substance.

FIG. 5A is a graph showing the amount of fibronectin determined in the culture supernatant of the HT/NEO cells, the HT/DN2 cells and the HT/DN14 cells with and without the addition of TGF-beta 1. The values represent the mean +/-S.D. of the amount of fibronectin in the culture supernatant prepared from three different wells. FIG. 5B is a graph showing the amount of fibronectin determined in the matrix extract of the HT/NEO cells, the HT/DN2 cells and the HT/DN14 cells with and without the addition of TGF-beta 1. The values represent the mean +/S.D. of the amount of fibronectin in the matrix extract prepared from three different wells.

FIG. 6A is a graph showing the amount of fibronectin determined in the culture supernatant of the MES/NEO cells, the MES/DN3 cells and the MES/DN6 cells with and without the addition of TGF-beta 1. The values represent the mean +/-S.D. of the amount of fibronectin in the culture supernatant prepared from three different wells. FIG. 6B is a graph showing the amount of fibronectin determined in the matrix extract of the MES/NEO cells, the MES/DN3 cells and the MES/DN6 cells with and without the addition of TGF-beta 1. The values represent the mean +/S.D. of the amount of fibronectin in the matrix extract prepared from three different wells.

FIG. 7 is a graph showing the amount of type I collagen determined in the culture supernatant of the MES/NEO cells, the MES/DN3 cells and the MES/DN6 cells with and without the addition of TGF-beta 1. The values represent the mean +/-S.D. of the amount of type I collagen in the culture supernatant prepared from three different wells.

FIG. 8 is a graph showing the amount of type IV collagen determined in the culture supernatant of the MES/NEO cells, the MES/DN3 cells and the MES/DN6 cells with and without the addition of TGF-beta 1. The values represent the mean +/-S.D. of the amount of type IV collagen in the culture supernatant prepared from three different wells.

FIG. 9 is a graph showing the result of a two-hybrid assay using the CHO cells. The values represent the mean +/-S.D. of the luciferase activity

in the culture supernatant prepared from three different wells.
FIG. 10 is a graph showing the amount of PAI-1 in the culture supernatant when TGF-beta 1 was added to the Mv1Lu cells. The values represent the mean +/-S.D. of the amount of PAI-1 in the culture supernatant prepared from three different wells.
FIG. 11 is the activity in Miller Units of beta-galactosidase of a yeast L40 that was transformed with an amino terminaltruncated TAB1 mutants (TAB1C45-TAB1C20) and the yeast 2-hybrid expression plasmid of TAK1. The measurement was conducted three times and the result is expressed in the mean +/-S.D. The values represent a ratio based on the beta-galactosidase activity of the yeast L40 that was transformed with TAB1C68 and the yeast 2-hybrid expression plasmid of TAK1.
FIG. 12 is the activity in Miller Units of beta-galactosidase of a yeast L40 that was transformed with a carboxy terminaltruncated TAB1 mutants (TAB1C45 Delta 14-TAB1C45 Delta 25) and a yeast 2-hybrid expression plasmid of TAK1. The measurement was conducted three times and the result is expressed in the mean +/-S.D. The values represent a ratio to the betagalactosidase activity of the yeast L40 that was transformed with TAB1C68 and the yeast 2-hybrid expression plasmid of TAK1.
FIG. 13A is the result of Western analysis of TAK1 and FLAG-TAB1 contained in the immunoprecipitate obtained using anti-TAK1 antibody in the presence or absence of each peptide. FIG. 13B is the result obtained by quantifying the density of bands each obtained by Western analysis and then by correcting the amount of the co-precipitated FLAG-TAB1 with the amount of TAK1. The values represent values relative to that obtained in the absence of the peptide which was set as 1.
FIG. 14 shows the ability of the TAB1 deletion mutants (TAB1C68, TAB1C45, TAB1C40, TAB1C35, TAB1C30 and TAB1C25) to bind to and activate TAK1.

AB A method for screening substances that **inhibit binding** between a **TAK1** polypeptide and a **TAB1** polypeptide, which comprises contacting the TAB1 polypeptide to the TAK1 polypeptide and a test sample and then detecting or determining the TAK1 polypeptide that is bound to the TAB1 polypeptide.

L6 ANSWER 3 OF 66 USPATFULL
AN 2002:221385 USPATFULL
TI TAB1 protein and DNA coding therefore
IN Matsuomoto, Kunihiro, Nagoya-shi, JAPAN
Nishida, Eisuke, Kyoto-shi, JAPAN
PA CHUGAI SEIYAKI KABUSHIKI KAISHA (non-U.S. corporation)
PI US 2002119525 A1 20020829
AI US 2002-123427 A1 20020417 (10)
RLI Division of Ser. No. US 2000-688701, filed on 17 Oct 2000, ABANDONED Division of Ser. No. US 1999-406854, filed on 29 Sep 1999, GRANTED, Pat. No. US 6140042 Division of Ser. No. US 1996-752891, filed on 20 Nov 1996, GRANTED, Pat. No. US 5837819
PRAI JP 1996-300856 19961028
JP 1996-126282 19960424
DT Utility
FS APPLICATION
LREP Stephen A. Bent, Foley & Lardner, Washington Harbour, Suite 500, 3000 K Street, N.W., Washington, DC, 20007-5143
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1057
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L6 ANSWER 4 OF 66 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE

3
AN 2002:630500 BIOSIS
DN PREV200200630500
TI TAK1-TAB1 fusion protein: A novel constitutively active mitogen-activated protein kinase kinase kinase that stimulates AP-1 and NF-kappaB signaling pathways.
AU Sakurai, Hiroaki; Nishi, Akito; Sato, Naoya; Mizukami, Junko; Miyoshi, Hidetaka; Sugita, Takahisa (1)
CS (1) Discovery Research Laboratory, Tanabe Seiyaku Co., Ltd., 16-89 Kashima 3-chome, Yodogawa-ku, Osaka, 532-8505: t-sugita@tanabe.co.jp Japan
SO Biochemical and Biophysical Research Communications, (October 11, 2002) Vol. 297, No. 5, pp. 1277-1281. http://www.academicpress.com/bbrc. print. ISSN: 0006-291X.
DT Article
LA English
AB TAK1 mitogen-activated protein kinase kinase kinase (MAP3K) is activated by its specific **activator, TAK1-binding** protein 1 (**TAB1**). A constitutively active **TAK1** mutant has not yet been generated due to the indispensable requirement of TAB1 for TAK1 kinase activity. In this study, we generated a novel constitutively active TAK1 by fusing its kinase domain to the minimal TAK1-activation domain of TAB1. Co-immunoprecipitation assay demonstrated that these domains interacted intra-molecularly. The TAK1-TAB1 fusion protein showed a significant MAP3K activity in vitro and activated c-Jun N-terminal kinase/p38 MAPKs and IkappaB kinase in vivo, which was followed by increased production of interleukin-6. These results indicate that the fusion protein is useful for characterizing the physiological roles of the TAK1-TAB1 complex.

L6 ANSWER 5 OF 66 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
AN 2000:278128 CAPLUS
DN 132:320956
TI Method for screening compound inhibiting signal transduction of inflammatory cytokine
IN Tsuchiya, Masayuki; Ohtomo, Toshihiko; Sugamata, Yasuhiro; Matsumoto, Kunihiro
PA Chugai Seiyaku K. K., Japan
SO PCT Int. Appl., 100 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000023610	A1	20000427	WO 1999-JP5817	19991021
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9962278	A1	20000508	AU 1999-62278	19991021
EP 1127944	A1	20010829	EP 1999-949347	19991021
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI JP 1998-299962	A	19981021		
WO 1999-JP5817	W	19991021		

AB By inhibiting the signal transduction of TAK1, effects of inflammatory cytokines are depressed, the prodn. of inflammatory cytokines (IL-1, TNF,

etc.) induced by inflammatory stimulus is depressed and the prodn. of other inflammatory cytokines (IL-6, etc.) induced by the inflammatory cytokines is depressed. The assay comprises contacting TAK1 and TAB1 (TAK1 kinase **binding** protein 1) with the sample, monitoring formation of **TAK1** kinase-**TAB1** complexes, and screening compd. that **inhibits TAK1-TAB1 binding**. The method may also use labeled anti-TAB1 antibody for drug screening.

RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 66 BIOTECHABS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2000-09271 BIOTECHABS
TI Method for screening inhibitors of TAK1 signal transduction for suppression of inflammatory cytokine production and use as antiinflammatory agents;
fusion protein and reporter gene assay for use in drug screening
AU Tsuchiya M; Ohtomo T; Sugamata Y; Matsumoto K
PA Chugai-Seiyaku
LO Tokyo, Japan.
PI WO 2000023610 27 Apr 2000
AI WO 1999-JP5817 21 Oct 1999
PRAI JP 1998-299962 21 Oct 1998
DT Patent
LA Japanese
OS WPI: 2000-339707 [29]
AB A new method for screening compounds for inhibition of inflammatory cytokine signal transduction is claimed and involves contacting the sample with **TAK1** and its receptor **TAB1** and selecting for inhibition of TAK/TAB1 **binding**. Also claimed is a method for screening compounds to **inhibit** inflammatory cytokine signal transduction in which the inhibition of TAK1 phosphorylation is selected for; and drug compositions for therapy of inflammatory disorders containing as active component an inflammatory cytokine signal transduction inhibitor. TAK1 is an essential component of the signalling process which results in release of inflammatory cytokines such as interleukin-1, interleukin-10, tumor necrosis factor and interleukin-6. The selection of effective antiinflammatory agents is possible. The TAK1 or TAB1 may be fused to another protein and/or immobilized on a support and labeled for an assay (sandwich immunoassay). The assay may also involve a reporter gene e.g. luciferase, green fluorescent protein, beta-galactosidase (EC-3.2.1.23) or chloramphenicol-acetyltransferase (EC-2.3.1.28). (100pp)

L6 ANSWER 7 OF 66 USPATFULL
AN 2000:146088 USPATFULL
TI TAB1 protein and DNA coding therefore
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Chugai Seiyaku Kabushiki Kaisha, Tokyo, Japan (non-U.S. corporation)
PI US 6140042 20001031
AI US 1999-406854 19990929 (9)
RLI Division of Ser. No. US 1996-752891, filed on 20 Nov 1996, now patented, Pat. No. US 5837819
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028
DT Utility
FS Granted
EXNAM Primary Examiner: Schwartzman, Robert A.; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner
CLMN Number of Claims: 1

ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1108
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L6 ANSWER 8 OF 66 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5
AN 1999:286157 CAPLUS
DN 130:334998
TI Method of screening TGF-.beta. inhibitory substances
IN Ono, Koichiro; Ohtomo, Toshihiko; Tsuchiya, Masayuki
PA Chugai Seiyaku Kabushiki Kaisha, Japan
SO PCT Int. Appl., 195 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9921010	A1	19990429	WO 1998-JP4796	19981022
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2306778	AA	19990429	CA 1998-2306778	19981022
	AU 9896468	A1	19990510	AU 1998-96468	19981022
	AU 752461	B2	20020919		
	EP 1043586	A1	20001011	EP 1998-950354	19981022
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	US 6451617	B1	20020917	US 2000-529279	20000411
	US 2002155624	A1	20021024	US 2002-158895	20020603
PRAI	JP 1997-290188	A	19971022		
	WO 1998-JP4796	W	19981022		
	US 2000-529279	A1	20000411		

AB A method of screening substances which **inhibit** the **binding** of TGF-.beta.-activated kinase 1 (**TAK1**) polypeptide to **TAK1-binding** protein (**TAB1**) polypeptide, characterized by contacting TAK1 polypeptide and a sample with TAB1 polypeptide and detecting or detg. the TAK1 polypeptide bonded to the TAB1 polypeptide. TAK1 and TAB1 polypeptides may be fusion proteins and may be labeled with radioisotope, enzyme or fluorescent substance for the screening assay. The TGF-.beta. inhibitor is TGF-.beta. signal transduction inhibitor, extracellular matrix protein prodn. inhibitor, cell proliferation inhibitor, monocyte migration inhibitor, physiol. active substance induction inhibitor, immunosuppression inhibitor, or amyloid .beta. protein pptn. inhibitor. Thus, human TAK1-6xHis, human TAB1-FLAG, and human MBP-TAB1C-FLAG fusion proteins were prepd., purified, and used together with anti-FLAG antibody in an ELISA.
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 66 CAPLUS COPYRIGHT 2002 ACS
AN 1999:511259 CAPLUS
DN 131:141477
TI NF-.kappa.B activation inhibitors, methods for screening the inhibitors

using the function of TGF-.beta. activated kinase 1 as parameter, and therapeutical use of the inhibitors for autoimmune diseases and inflammation

IN Sugita, Takahisa; Sakurai, Hiroaki; Kageyama, Noriko; Hasegawa, Ko

PA Tanabe Seiyaku Co., Ltd., Japan

SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2

DT Patent

LA Japanese

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9940202	A1	19990812	WO 1999-JP422	19990202
W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GD, GE, HR, HU, ID, IL, IN, IS, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9920764	A1	19990823	AU 1999-20764	19990202
JP 2000197500	A2	20000718	JP 1999-26803	19990204
PRAI JP 1998-26003	A	19980206		
JP 1998-309316	A	19981030		
WO 1999-JP422	W	19990202		

AB Described is a method of identifying nuclear factor .kappa.B (NF-.kappa.B) activation inhibitors, which have prophylactic and therapeutic uses for autoimmune diseases and inflammation, by testing whether a sample substance is able to inhibit the function of TGF-.beta. activated kinase 1 (TAK1). The function of TAK1 is selected from (1) interaction between TAK1 and TAK1-binding protein 1 (TAB1); (2) protein kinase activity of TAK1; (3) TAK1-mediated intracellular activation of the I.kappa.B kinase (IKK) complex; and (4) TAK1-mediated NF-.kappa.B activation. The method was demonstrated using a yeast two-hybrid system (using the TAK1-TAB1 interaction as a marker and .beta.-galactosidase a reporter).

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 66 CAPLUS COPYRIGHT 2002 ACS

AN 1999:752266 CAPLUS

DN 132:10515

TI Substances which inhibit binding of specific proteins to XIAP, screening of them, and their use as drugs

IN Matsumoto, Kunihiro

PA Japan

SO Jpn. Kokai Tokkyo Koho, 43 pp.
CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 11326328	A2	19991126	JP 1998-130378	19980513

AB Substances which **inhibit binding** of **TAB1** [**TAK1 binding** protein 1 (**TAK1**: TGF-.beta.-activated kinase 1)], TGF-.beta. type I receptor (T.beta.R-I), or TGF-.beta. type II receptor (T.beta.R-II) to XIAP (X-linked inhibitor of apoptosis protein) are screened by examg. whether or not XIAP binds to them when XIAP are contacted with TAB1, T.beta.R-I, or T.beta.R-II and a sample to be tested. XIAP binding is examd. by detecting TAB1 or XIAP-mediated biol. activity of TGF-.beta., e.g. change in expression of reporter genes, e.g. for PAI-1, fibronectin, and type I and IV collagens.

The substances inhibit or activate the biol. phenomena, e.g. acceleration of extracellular matrix protein prodn., cell proliferation inhibition, monocyte migration, bioactive substance induction, immunosuppression, and .beta.-amyloid deposition, through blocking TGF-.beta. signaling, and are useful for treatment of liver fibrosis, lung fibrosis, glomerulonephritis, diabetic nephropathy, nephrosclerosis, vascular restenosis, keloid, scleroderma, autoimmune diseases, and Alzheimer disease. Cloning of some XIAP proteins (IAP family) functioning upstream of TAB1-TAK1 using yeast two-hybrid system, binding specificity of TAB1 to XIAP, interaction interaction between XIAP and TGF-.beta. type I and II receptors, and effect of XIAP on TGF-.beta. signaling were shown.

L6 ANSWER 11 OF 66 TOXCENTER COPYRIGHT 2002 ACS
AN 1999:210326 TOXCENTER
CP Copyright 2002 ACS
DN CA13202010515E
TI Substances which inhibit binding of specific proteins to XIAP, screening of them, and their use as drugs
AU Matsumoto, Kunihiro
PI JP 99326328 A2 26 Nov 1999
SO (1999) Jpn. Kokai Tokkyo Koho, 43 pp.
CODEN: JKXXAF.
CY JAPAN
DT Patent
FS CAPLUS
OS CAPLUS 1999:752266
LA Japanese
ED Entered STN: 20011116
Last Updated on STN: 20020403
AB Substances which **inhibit binding** of **TAB1** [**TAK1 binding** protein 1 (**TAK1**: TGF-.beta.-activated kinase 1)], TGF-.beta. type I receptor (T.beta.R-I), or TGF-.beta. type II receptor (T.beta.R-II) to XIAP (X-linked inhibitor of apoptosis protein) are screened by examg. whether or not XIAP binds to them when XIAP are contacted with TAB1, T.beta.R-I, or T.beta.R-II and a sample to be tested. XIAP binding is examd. by detecting TAB1 or XIAP-mediated biol. activity of TGF-.beta., e.g. change in expression of reporter genes, e.g. for PAI-1, fibronectin, and type I and IV collagens. The substances inhibit or activate the biol. phenomena, e.g. acceleration of extracellular matrix protein prodn., cell proliferation inhibition, monocyte migration, bioactive substance induction, immunosuppression, and .beta.-amyloid deposition, through blocking TGF-.beta. signaling, and are useful for treatment of liver fibrosis, lung fibrosis, glomerulonephritis, diabetic nephropathy, nephrosclerosis, vascular restenosis, keloid, scleroderma, autoimmune diseases, and Alzheimer disease. Cloning of some XIAP proteins (IAP family) functioning upstream of TAB1-TAK1 using yeast two-hybrid system, binding specificity of TAB1 to XIAP, interaction interaction between XIAP and TGF-.beta. type I and II receptors, and effect of XIAP on TGF-.beta. signaling were shown.

L6 ANSWER 12 OF 66 USPATFULL
AN 1999:150965 USPATFULL
TI Tab1 protein and DNA coding therefor
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Chugai Seiyaku Kabushiki Kaisha, Tokyo, Japan (non-U.S. corporation)
PI US 5989862 19991123
AI US 1998-144178 19980831 (9)
RLI Division of Ser. No. US 1996-752891, filed on 20 Oct 1996, now patented, Pat. No. US 5837819
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028

DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1049
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L6 ANSWER 13 OF 66 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 6
AN 1999:236235 BIOSIS
DN PREV199900236235
TI Functional interactions of transforming growth factor beta-activated kinase 1 with IkappaB kinases to stimulate NF-kappaB activation.
AU Sakurai, Hiroaki; Miyoshi, Hidetaka; Toriumi, Wataru; Sugita, Takahisa (1)
CS (1) Discovery Research Laboratory, Tanabe Seiyaku Co., Ltd., 16-89 Kashima 3-chome, Yodogawa-ku, Osaka, 532-8505 Japan
SO Journal of Biological Chemistry, (April 9, 1999) Vol. 274, No. 15, pp. 10641-10648.
ISSN: 0021-9258.
DT Article
LA English
SL English
AB Several mitogen-activated protein kinase kinase kinases play critical roles in nuclear factor-kappaB (NF-kappaB) activation. We recently reported that the overexpression of transforming growth factor-beta-activated kinase 1 (TAK1), a member of the mitogen-activated protein kinase kinase kinase family, together with its **activator TAK1-binding** protein 1 (**TAB1**) stimulates NF-kappaB activation. Here we investigated the molecular mechanism of TAK1-induced NF-kappaB activation. Dominant negative mutants of IkappaB kinase (IKK) alpha and IKKbeta inhibited TAK1-induced NF-kappaB activation. TAK1 activated IKKalpha and IKKbeta in the presence of TAB1. IKKalpha and IKKbeta were coimmunoprecipitated with TAK1 in the absence of TAB1. TAB1-induced TAK1 activation promoted the dissociation of active forms of IKKalpha and IKKbeta from active TAK1, whereas the IKK mutants remained to interact with active TAK1. Furthermore, tumor necrosis factor-alpha activated endogenous TAK1, and the kinase-negative TAK1 acted as a dominant negative inhibitor against tumor necrosis factor-alpha-induced NF-kappaB activation. These results demonstrated a novel signaling pathway to NF-kappaB activation through TAK1 in which TAK1 may act as a regulatory kinase of IKKs.

L6 ANSWER 14 OF 66 USPATFULL
AN 1998:144215 USPATFULL
TI TAB1 protein
IN Matsuomoto, Kunihiro, Nagoya, Japan
Nishida, Eisuke, Kyoto, Japan
PA Ueno, Naoto, Sapporo, Japan (non-U.S. individual)
PI US 5837819 19981117
AI US 1996-752891 19961120 (8)
PRAI JP 1996-126282 19960424
JP 1996-300856 19961028
DT Utility
FS Granted
EXNAM Primary Examiner: Eliott, George C.; Assistant Examiner: McGarry, Sean
LREP Foley & Lardner

CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 910
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB TAB1 protein having activity which activates factor TAK1 in the TGF-.beta. signaling pathway, and having the amino acid sequence shown in FIG. 1.

L6 ANSWER 15 OF 66 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7
AN 1998:164435 BIOSIS
DN PREV199800164435
TI TGF-beta-activated kinase 1 stimulates NF-kappaB activation by an NF-kappaB-inducing kinase-independent mechanism.
AU Sakurai, Hiroaki; Shigemori, Noriko; Hasegawa, Ko; Sugita, Takahisa (1)
CS (1) Lead Generation Res. Lab., Tanabe Seiyaku Co. Ltd., 16-89 Kashima 3-chome, Yodogawa-ku, Osaka 532-0031 Japan
SO Biochemical and Biophysical Research Communications, (Feb. 13, 1998) Vol. 243, No. 2, pp. 545-549.
ISSN: 0006-291X.
DT Article
LA English
AB Several mitogen-activated protein kinase kinase kinases (MAPKKKs), including NF-kappaB-inducing kinase (NIK), play critical roles in NF-kappaB activation. We isolated cDNA for human TGF-beta activated kinase 1 (TAK1), a member of the MAPKKK family, and evaluated its ability to stimulate NF-KAPPAB activation. Overexpression of TAK1 together with its **activator** protein, **TAK1 binding** protein 1 (**TAB1**), induced the nuclear translocation of NF-kappaB p50/p65 heterodimer accompanied by the degradation of IkappaBalpha and IkappaBbeta and the expression of independent reporter gene. A dominant negative mutant of NIK did not inhibit TAK1-induced NF-kappaB activation. These results suggest that TAK1 induces NF-kappaB activation through a novel NIK-independent signaling pathway.

L6 ANSWER 16 OF 66 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 8
AN 1997:720160 CAPLUS
DN 128:11258
TI TAB1 protein and its variant and gene structure
IN Matsumoto, Kunihiro; Nishida, Elsuke
PA Ueno, Naoto, Japan
SO Eur. Pat. Appl., 30 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 803571	A2	19971029	EP 1997-302808	19970424
	EP 803571	A3	19990728		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 10004976	A2	19980113	JP 1996-300856	19961028
	US 5837819	A	19981117	US 1996-752891	19961120
	US 5989862	A	19991123	US 1998-144178	19980831
	US 6140042	A	20001031	US 1999-406854	19990929
	US 2002119525	A1	20020829	US 2002-123427	20020417
PRAI	JP 1996-126282	A	19960424		
	JP 1996-300856	A	19961028		
	US 1996-752891	A	19961120		
	US 1999-406854	A3	19990929		

US 2000-688701 B3 20001017
AB Claims include the DNA coding for **TAB1** (protein **TAK1** kinase-**binding**) protein having activity which **activates** factor TAK1 in the TGF-.beta. signaling pathway, and the amino acid sequence of TAB1. TGF-.beta. formation in cells was induced by protein TAB1, TAK1 (kinase), and the combination of TAB1 and TAK1.

L6 ANSWER 17 OF 66 CAPLUS COPYRIGHT 2002 ACS
AN 1996:715668 CAPLUS
DN 126:43981
TI MAP kinase kinase kinase, TAK1 functions in TGF-.beta.-mediated signal transduction
AU Irie, Kenji; Shibuya, Hiroshi
CS Fac. Sci., Nagoya Univ., Nagoya, 464-01, Japan
SO Jikken Igaku (1996), 14(19), 2616-2622
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; General Review
LA Japanese
AB A review with 21 refs., on isolation and function of a new TAK1 kinase and its **activator**, **TAK1-binding** protein 1 (**TAB1**), in TGF-.beta.-mediated signal transduction pathway. Involvement of TAK1 and TAB1 in TGF-.beta.-stimulated PAI-1 gene expression is also discussed.

L6 ANSWER 18 OF 66 CAPLUS COPYRIGHT 2002 ACS
AN 1996:319410 CAPLUS
DN 125:82669
TI TAB1: an activator of the TAK1 MAPKKK in TGF-.beta. signal transduction
AU Shibuya, Hiroshi; Yamaguchi, Kyoko; Shirakabe, Kyoko; Tonegawa, Akane; Gotoh, Yukiko; Ueno, Naoto; Irie, Kenji; Nishida, Eisuke; Matsumoto, Kunihiro
CS Faculty Pharmaceutical Sciences, Hokkaido Univ., Sapporo, 060, Japan
SO Science (Washington, D. C.) (1996), 272(5265), 1179-1182
CODEN: SCIEAS; ISSN: 0036-8075
PB American Association for the Advancement of Science
DT Journal
LA English
AB Transforming growth factor-.beta. (TGF-.beta.) regulates many aspects of cellular function. A member of the mitogen-activated protein kinase kinase kinase (MAPKKK) family, TAK1, was previously identified as a mediator in the signaling pathway of TGF-.beta. superfamily members. The yeast two-hybrid system has now revealed 2 human proteins, termed TAB1 and TAB2 (for TAK1 binding protein), that interact with TAK1. TAB1 and TAK1 were co-immunopptd. from mammalian cells. Ovrprodn. of TAB1 enhanced activity of the plasminogen activator inhibitor 1 gene promoter, which is regulated by TGF-.beta., and increased the kinase activity of TAK1. TAB1 may function as an activator of the TAK1 MAPKKK in TGF-.beta. signal transduction.

L6 ANSWER 19 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09544 peptide DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]

AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a peptide from an example of the present invention.

L6 ANSWER 20 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09543 peptide DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a peptide from an example of the present invention.

L6 ANSWER 21 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09542 Protein DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of

bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents human TAK1.

L6 ANSWER 22 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09541 Protein DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents human TAB1.

L6 ANSWER 23 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09547 Protein DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or

activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents TAK1-6xHis from an example of the present invention.

L6 ANSWER 24 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09546 Protein DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents TAB1-FLAG from an example of the present invention.

L6 ANSWER 25 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09545 peptide DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase

activity. The present sequence represents a peptide from an example of the present invention.

L6 ANSWER 26 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09548 peptide DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a peptide from an example of the present invention.

L6 ANSWER 27 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09550 Protein DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents human TAB1.

L6 ANSWER 28 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAY09549 peptide DGENE

TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a peptide from an example of the present invention.

L6 ANSWER 29 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56281 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 30 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56280 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.

PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 31 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56279 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence encodes human TAK1.

L6 ANSWER 32 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56294 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese

OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 33 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56293 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 34 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56292 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the

polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 35 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56291 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 36 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56290 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal

transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 37 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56289 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 38 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56288 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators,

or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 39 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56287 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 40 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56286 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in

an example from the present invention.

L6 ANSWER 41 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56285 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence encodes TAK1-6xHis from an example of the present invention.

L6 ANSWER 42 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56284 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 43 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56283 DNA DGENE

TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 44 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56282 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence encodes TAB1-FLAG from an example of the present invention.

L6 ANSWER 45 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56309 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.

PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 46 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56308 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 47 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56307 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent

LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 48 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56306 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 49 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56305 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to

TAB1 polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 50 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56304 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 51 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56303 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory

substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 52 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56302 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 53 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56301 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or

activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 54 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56300 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 55 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56299 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase

activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 56 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56298 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 57 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56297 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 58 OF 66 DGENE (C) 2002 THOMSON DERWENT

AN AAX56296 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 59 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56295 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 60 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56278 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M

PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence encodes human TAB1.

L6 ANSWER 61 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56314 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 62 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56313 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent

LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 63 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56315 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to **TAB1** polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 64 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56312 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
IN Ohtomo T; Ono K; Tsuchiya M
PA (CHUS) CHUGAI SEIYAKU KK.
PI WO 9921010 A1 19990429 195p
AI WO 1998-JP4796 19981022
PRAI JP 1997-290188 19971022
DT Patent
LA Japanese
OS 1999-312645 [26]
AB A method has been developed for screening for substances which **inhibit** the **binding** of **TAK1** polypeptide to

TAB1 polypeptide. The method comprises: (a) contacting the polypeptide in the presence of a sample; and (b) detecting the amount of bound polypeptide, in which the sample can be pre-mixed with TAK1 or TAB1 polypeptide first. The transforming growth factor (TGF)-beta inhibitory substances can be used in drugs for indications e.g. as TGF-beta signal transmission inhibitors or activators, or extracellular matrix protein production enhancement inhibitors or activators, or cell proliferation prevention inhibitors or activators, or monocyte migration inhibitors or activators, or physiological activity induction inhibitors or activators, or immunosuppression inhibitors or activators, or amyloid beta protein precipitation inhibitors or activators, and such substances can also be inhibitors of the TAK1 polypeptide function, particularly kinase activity. The present sequence represents a PCR primer which is used in an example from the present invention.

L6 ANSWER 65 OF 66 DGENE (C) 2002 THOMSON DERWENT
AN AAX56311 DNA DGENE
TI Screening for TGF- beta inhibitory substances, which are useful as drugs for treatment of diseases relating to its disorder
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NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
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NEWS 19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
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NEWS 24	Sep 16	Experimental properties added to the REGISTRY file
NEWS 25	Sep 16	Indexing added to some pre-1967 records in CA/CAPLUS
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=> s tgf beta activated kinase
  17 FILES SEARCHED...
  36 FILES SEARCHED...
  56 FILES SEARCHED...
  85 FILES SEARCHED...
L1          316 TGF BETA ACTIVATED KINASE

=> s transforming growth factor beta activated kinase
  12 FILES SEARCHED...
  23 FILES SEARCHED...
  38 FILES SEARCHED...
  47 FILES SEARCHED...
  60 FILES SEARCHED...
  85 FILES SEARCHED...
L2          186 TRANSFORMING GROWTH FACTOR BETA ACTIVATED KINASE

=> s l1 or l2
  58 FILES SEARCHED...
L3          436 L1 OR L2

=> s l3 (3A) (inhibitor or antagonist)
  25 FILES SEARCHED...
  49 FILES SEARCHED...
  87 FILES SEARCHED...
L4            3 L3 (3A) (INHIBITOR OR ANTAGONIST)

=> s l4 and cytokine
  39 FILES SEARCHED...
  85 FILES SEARCHED...
L5            0 L4 AND CYTOKINE

=> s l3 (s) cytokine
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L221 (S) CYTOKINE'
  31 FILES SEARCHED...
  40 FILES SEARCHED...
  81 FILES SEARCHED...
L6           37 L3 (S) CYTOKINE

=> s l3 (25A) cytokine
  15 FILES SEARCHED...
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  47 FILES SEARCHED...
  67 FILES SEARCHED...
L7           0 L3 (25A) CYTOKINE

=> s l3 and cytokine
  39 FILES SEARCHED...
  68 FILES SEARCHED...
 75% OF LIMIT FOR L#S REACHED
L8          62 L3 AND CYTOKINE

=> s l8 and (inhibitor or antagonist)
SEARCH FILE UNAVAILABLE FOR DGENE
  29 FILES SEARCHED...
  55 FILES SEARCHED...
  78 FILES SEARCHED...
L9          15 L8 AND (INHIBITOR OR ANTAGONIST)

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L10           8 DUPLICATE REMOVE L9 (7 DUPLICATES REMOVED)

=> d l10 1-8 bib ab

L10  ANSWER 1 OF 8  USPATFULL
AN     2002:199273  USPATFULL
TI     Novel human kinase and polynucleotides encoding the same
IN     Hu, Yi, Spring, TX, UNITED STATES
       Kieke, James Alvin, Houston, TX, UNITED STATES
       Donoho, Gregory, Portage, MI, UNITED STATES
PI     US 2002107384      A1   20020808
AI     US 2001-14882      A1   20011211 (10)
PRAI   US 2000-254744P     20001211 (60)
DT     Utility
FS     APPLICATION
LREP   LEXICON GENETICS INCORPORATED, 8800 TECHNOLOGY FOREST PLACE, THE
       WOODLANDS, TX, 77381-1160
CLMN   Number of Claims: 2
ECL    Exemplary Claim: 1
DRWN   No Drawings
LN.CNT 1246
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB     Novel human polynucleotide and polypeptide sequences are disclosed that
       can be used in therapeutic, diagnostic, and pharmacogenomic
       applications.

L10  ANSWER 2 OF 8  USPATFULL
AN     2002:191541  USPATFULL
TI     Protein-protein interactions
IN     Heichman, Karen, Salt Lake City, UT, UNITED STATES
       Bartel, Paul L., Salt Lake City, UT, UNITED STATES
PI     US 2002102606      A1   20020801
```

AI US 2001-847599 A1 20010503 (9)
PRAI US 2000-201722P 20000504 (60)
DT Utility
FS APPLICATION
LREP ROTHWELL, FIGG, ERNST & MANBECK, P.C., 555 13TH STREET, N.W., SUITE 701, EAST TOWER, WASHINGTON, DC, 20004
CLMN Number of Claims: 40
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1917
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to the discovery of novel protein-protein interactions that are involved in mammalian physiological pathways, including physiological disorders or diseases. Examples of physiological disorders and diseases include non-insulin dependent diabetes mellitus (NIDDM), neurodegenerative disorders, such as Alzheimer's Disease (AD), and the like. Thus, the present invention is directed to complexes of these proteins and/or their fragments, antibodies to the complexes, diagnosis of physiological generative disorders (including diagnosis of a predisposition to and diagnosis of the existence of the disorder), drug screening for agents which modulate the interaction of proteins described herein, and identification of additional proteins in the pathway common to the proteins described herein.

L10 ANSWER 3 OF 8 USPATFULL
AN 2002:60946 USPATFULL
TI Novel human protein kinases and uses therefor
IN Meyers, Rachel, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PA Millennium Pharmaceuticals, Inc. (U.S. corporation)
PI US 2002034780 A1 20020321
AI US 2001-799875 A1 20010306 (9)
RLI Continuation-in-part of Ser. No. US 2000-659287, filed on 12 Sep 2000, PENDING
PRAI US 2000-182059P 20000211 (60)
DT Utility
FS APPLICATION
LREP ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 58 Drawing Page(s)
LN.CNT 6018
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel kinase nucleic acid sequences and proteins. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

L10 ANSWER 4 OF 8 USPATFULL
AN 2002:12239 USPATFULL
TI Methods for using 20893, a human protein kinase
IN Galvin, Katherine M., Jamaica Plain, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Weich, Nadine S., Brookline, MA, UNITED STATES
PI US 2002006618 A1 20020117
AI US 2001-780949 A1 20010209 (9)
PRAI US 2000-181690P 20000209 (60)
DT Utility
FS APPLICATION
LREP ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000

CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 23 Drawing Page(s)
LN.CNT 4723
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods for using a human protein kinase belonging to the superfamily of mammalian protein kinases. The invention also relates to methods for using polynucleotides encoding the protein kinase. The invention relates to methods using the protein kinase polypeptides and polynucleotides as a target for diagnosis and treatment in protein kinase-mediated or -related disorders. The invention further relates to drug-screening methods using the protein kinase polypeptides and polynucleotides to identify agonists and **antagonists** for diagnosis and treatment. The invention further encompasses agonists and **antagonists** based on the protein kinase polypeptides and polynucleotides. The invention further relates to agonists and **antagonists** identified by drug screening methods with the protein kinase polypeptides and polynucleotides as a target.

L10 ANSWER 5 OF 8 USPATFULL
AN 2002:29278 USPATFULL
TI Antisense inhibition of HPK/GCK-like kinase expression
IN Dean, Nicholas M., Olivenhain, CA, United States
Cowsert, Lex M., Carlsbad, CA, United States
PA ISIS Pharmaceuticals, Inc., Carlsbad, CA, United States (U.S. corporation)
PI US 6346416 B1 20020212
AI US 2000-651011 20000829 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: McGarry, Sean; Assistant Examiner: Lacourciere, Karen A
LREP Licata & Tyrrell P.C.
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 3123
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense compounds, compositions and methods are provided for modulating the expression of HPK/GCK-like kinase. The compositions comprise antisense compounds, particularly antisense oligonucleotides, targeted to nucleic acids encoding HPK/GCK-like kinase. Methods of using these compounds for modulation of HPK/GCK-like kinase expression and for treatment of diseases associated with expression of HPK/GCK-like kinase are provided.

L10 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2002:202327 BIOSIS
DN PREV200200202327
TI Transforming growth factor-beta1 transcriptionally activates CD34 and prevents induced differentiation of TF-1 cells in the absence of any cell-cycle effects.
AU Marone, M. (1); Scambia, G.; Bonanno, G.; Rutella, S.; de Ritis, D.; Guidi, F.; Leone, G.; Pierelli, L.
CS (1) Inst of Gynecology, Catholic University, Largo A Gemelli 8, 00168, Rome Italy
SO Leukemia (Basingstoke), (January, 2002) Vol. 16, No. 1, pp. 94-105. print. ISSN: 0887-6924.
DT Article
LA English
AB A number of **cytokines** modulate self-renewal and differentiation

of hematopoietic elements. Among these is transforming growth factor beta1 (TGF-beta1), which regulates cell cycle and differentiation of hematopoietic cells, but has pleiotropic activities depending on the state of responsiveness of the target cells. It has been previously shown by us and other authors that TGF-beta1 maintains human CD34+ hematopoietic progenitors in an undifferentiated state, independently of any cell cycle effects, and that depletion of TGF-beta1 triggers differentiation accompanied by a decrease in CD34 antigen expression. In the present work, we show that exogenous TGF-beta1 upregulates the human CD34 antigen in the CD34+ cell lines TF-1 and KG-1a, but not in the more differentiated CD34- cell lines HL-60 and K-562. We further studied this effect in the pluripotent erythroleukemia cell line TF-1. Here, TGF-beta1 did not effect cell growth, but induced transcriptional activation of full-length CD34 and prevented differentiation induced by differentiating agents. This effect was associated with nuclear translocation of Smad-2, activation of TAK-1, and with a dramatic decrease in p38 phosphorylation. In other systems TGF-beta1 has been shown to activate a **TGF-beta -activated kinase** 1 (TAK1), which in turn, activates p38. The specific **inhibitor** of p38 phosphorylation, SB202190, also increased CD34 RNA expression, indicating the existence of a link between p-38 inhibition by TGF-beta1 and CD34 overexpression. Our data demonstrate that TGF-beta1 transcriptionally activates CD34 and prevents differentiation of TF-1 cells by acting independently through the Smad, TAK1 and p38 pathways, and thus provide important clues for the understanding of hematopoietic development and a potential tool to modify response of hematopoietic cells to mitogens or differentiating agents.

L10 ANSWER 7 OF 8 USPATFULL
AN 2001:231143 USPATFULL
TI Arrays for identifying agents which mimic or inhibit the activity of interferons
IN Silverman, Robert H., Beachwood, OH, United States
Williams, Bryan R. G., Cleveland, OH, United States
Der, Sandy, Cleveland, OH, United States
PA The Cleveland Clinic Foundation, Cleveland, OH, United States (U.S. corporation)
PI US 6331396 B1 20011218
AI US 1999-405438 19990923 (9)
PRAI US 1998-101497P 19980923 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Zitomer, Stephanie; Assistant Examiner: Forman, B J
LREP Calfee, Halter & Griswold LLP
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 9639
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and model systems for identifying and characterizing new therapeutic agents, particularly proteins, which mimic or inhibit the activity of all interferons, Type I interferons, IFN-.alpha., IFN-.beta., or IFN-.gamma.. The method comprises administering an interferon selected from the group consisting of IFN-.alpha., IFN .beta., IFN-.tau., IFN-.omega., IFN-.gamma., and combinations thereof to cultured cells, administering the candidate agent to a duplicate culture of cells; and measuring the effect of the candidate agent and the interferon on the transcription or translation of one or, preferably, a plurality of the interferon stimulated genes or the interferon repressed genes (hereinafter referred to as "ISG's" and "IRGs", respectively). The model system is an array with gene probes that hybridize with from about 100 to about 5000 ISG and IRG transcripts.

L10 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
AN 2001:761049 CAPLUS
DN 136:52546
TI Raf kinase **inhibitor** protein interacts with NF-.kappa.B-inducing kinase and TAK1 and inhibits NF-.kappa.B activation
AU Yeung, Kam C.; Rose, David W.; Dhillon, Amardeep S.; Yaros, Diane; Gustafsson, Marcus; Chatterjee, Devasis; McFerran, Brian; Wyche, James; Kolch, Walter; Sedivy, John M.
CS Department of Molecular Biology, Cell Biology, Brown University, Providence, RI, 02912, USA
SO Molecular and Cellular Biology (2001), 21(21), 7207-7217
CODEN: MCEBD4; ISSN: 0270-7306
PB American Society for Microbiology
DT Journal
LA English
AB The Raf kinase **inhibitor** protein (RKIP) acts as a neg. regulator of the mitogen-activated protein (MAP) kinase (MAPK) cascade initiated by Raf-1. RKIP inhibits the phosphorylation of MAP/extracellular signal-regulated kinase 1 (MEK1) by Raf-1 by disrupting the interaction between these two kinases. The authors show here that RKIP also antagonizes the signal transduction pathways that mediate the activation of the transcription factor nuclear factor kappa B (NF-.kappa.B) in response to stimulation with tumor necrosis factor .alpha. (TNF-.alpha.) or interleukin 1.beta.. Modulation of RKIP expression levels affected NF-.kappa.B signaling independent of the MAPK pathway. Genetic epistasis anal. involving the ectopic expression of kinases acting in the NF-.kappa.B pathway indicated that RKIP acts upstream of the kinase complex that mediates the phosphorylation and inactivation of the **inhibitor** of NF-.kappa.B (I.kappa.B). In vitro kinase assays showed that RKIP antagonizes the activation of the I.kappa.B kinase (IKK) activity elicited by TNF-.alpha.. RKIP phys. interacted with 4 kinases of the NF-.kappa.B activation pathway, NF-.kappa.B-inducing kinase, **transforming growth factor .beta.-activated kinase** 1 (TAK1), IKK.alpha., and IKK.beta.. This mode of action bears striking similarities to the interactions of RKIP with Raf-1 and MEK1 in the MAPK pathway. Emerging data from diverse organisms suggest that RKIP and RKIP-related proteins represent a new and evolutionarily highly conserved family of protein kinase regulators. Since the MAPK and NF-.kappa.B pathways have physiol. distinct roles, the function of RKIP may be, in part, to coordinate the regulation of these pathways.
RE.CNT 79 THERE ARE 79 CITED REFERENCES AVAILABLE FOR THIS RECORD
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7	4830	tgf adj (beta or b)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:45
13	6128	(transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:46
19	16	((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) adj activated adj kinase	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:47
25	0	(((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) adj activated adj kinase) near10 cytokine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:48
31	0	(((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) adj activated adj kinase) near10 inflammatory	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:48
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43	0	(((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) adj activated adj kinase) near10 (il-1 or tnf)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:54
49	683	((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) near10 (inflammatory or cytokine)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:54
55	46	((transforming adj growth adj factor adj (beta or B)) or (tgf adj (beta or b))) near10 (inflammatory adj cytokine)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/12/18 13:55

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NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
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NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
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NEWS 29	Oct 24	BEILSTEIN adds new search fields
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NEWS 33	Nov 25	More calculated properties added to REGISTRY
NEWS 34	Dec 02	TIBKAT will be removed from STN
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=> s (transforming growth factor b) or (tgf b)
  11 FILES SEARCHED...
  22 FILES SEARCHED...
  30 FILES SEARCHED...
  43 FILES SEARCHED...
  55 FILES SEARCHED...
  69 FILES SEARCHED...
  84 FILES SEARCHED...
L1        2455 (TRANSFORMING GROWTH FACTOR B) OR (TGF B)

=> s (transforming growth factor beta) or (tgf beta)
  11 FILES SEARCHED...
  19 FILES SEARCHED...
  29 FILES SEARCHED...
  41 FILES SEARCHED...
  49 FILES SEARCHED...
  60 FILES SEARCHED...
  81 FILES SEARCHED...
  91 FILES SEARCHED...
L2      227388 (TRANSFORMING GROWTH FACTOR BETA) OR (TGF BETA)

=> l1 or l2
L1 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l1 or l2
  33 FILES SEARCHED...
  68 FILES SEARCHED...
  89 FILES SEARCHED...
L3      228440 L1 OR L2

=> s l3 (10a) (inflammatory cytokine)
  14 FILES SEARCHED...
  42 FILES SEARCHED...
  70 FILES SEARCHED...
L4        1399 L3 (10A) (INFLAMMATORY CYTOKINE)
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=> s l2 w (activated kinase)
MISSING OPERATOR L2 W
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l2 (w) (activated kinase)
  29 FILES SEARCHED...
  55 FILES SEARCHED...
  91 FILES SEARCHED...
L5          457 L2 (W) (ACTIVATED KINASE)

=> s l5 and l4
  55 FILES SEARCHED...
L6            0 L5 AND L4

=> s l4 and review
  39 FILES SEARCHED...
  70 FILES SEARCHED...
L7          154 L4 AND REVIEW

=> s l7 and PY<=1999
'1999' NOT A VALID FIELD CODE
   4 FILES SEARCHED...
   7 FILES SEARCHED...
   9 FILES SEARCHED...
  12 FILES SEARCHED...
  14 FILES SEARCHED...
  17 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
  29 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
  39 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
  45 FILES SEARCHED...
  47 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
  52 FILES SEARCHED...
  55 FILES SEARCHED...
  60 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
  68 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
  74 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
  82 FILES SEARCHED...
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
'1999' NOT A VALID FIELD CODE
 75% OF LIMIT FOR L#S REACHED
L8           55 L7 AND PY<=1999

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L9 ANSWER 1 OF 25 USPATFULL
AN 2002:246721 USPATFULL
TI Methods and materials for treating inflammatory diseases
IN Goronzy, Jorg J., Rochester, MN, United States
Weyand, Cornelia M., Rochester, MN, United States
PA Mayo Foundation for Medical Education and Research, Rochester, MN, United States (U.S. corporation)
PI US 6455497 B1 20020924
WO 9948514 19990930 <--
AI US 2000-646757 20001130 (9)
WO 1999-US6576 19990325
20001120 PCT 371 date
DT Utility
FS GRANTED
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Fish & Richardson, P.C. P.A.
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1254
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides methods and materials related to the treatment of inflammatory diseases such as rheumatoid arthritis. Specifically, the invention provides methods and materials for treating inflammation by reducing production of an inflammatory cytokine such as IFN-.gamma., IL-, and TNF-.alpha.. The invention also provides methods and materials for identifying reagents that can be used to treat inflammatory diseases. Specifically, the invention provides non-human animals containing human synovial tissue as well as methods for using such non-human animals to determine the influence of various test reagents on the inflamed state of human synovial tissue.

L9 ANSWER 2 OF 25 DDFU COPYRIGHT 2002 THOMSON DERWENT
AN 1999-40702 DDFU T S
TI Cytokines and mediators as therapeutic agents.
AU Lorenz H M; Kalden J R
CS Univ.Erlangen
LO Erlangen, Ger.
SO Internist (40, No. 9, 945-50, 1999) 30 Ref.
CODEN: INTEAG ISSN: 0020-9554
AV Medizinische Klinik III mit Poliklinik, Friedrich-Alexander Universitaet Erlangen-Nuernberg, Krankenhausstrasse 12, D-91054 Erlangen, Germany.
LA German
DT Journal
FA AB; LA; CT
FS Literature
AB This **review** deals with the importance of cytokines and mediators in the pathogenesis and possible treatment of rheumatoid arthritis (RA). Anti-**inflammatory cytokines** such as IL-4, IL-19, IL-13 and **TGF-beta** are ineffective in

preventing or influencing the inflammatory process. The possibilities of using monoclonal antibodies against TNF-alpha, IL-1 and IL-6 are currently being explored and results with those against TNF-alpha are the most promising. IL-6 antibodies are about to be tested. The antiviral IFN-gamma has proved less effective than TNF-alpha monoclonal antibodies, can trigger serum anti-DNA antibodies and is no longer used in RA.

L9 ANSWER 3 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
AN 1999:522070 CAPLUS
DN 132:62687
TI Laureate ESCI award for excellence in clinical science 1999. Cytokines and the human immunodeficiency virus: from bench to bedside
AU Poli, G.
CS San Raffaele Scientific Institute, Milan, 20132, Italy
SO European Journal of Clinical Investigation (**1999**), 29(8), 723-732
CODEN: EJCIB8; ISSN: 0014-2972
PB Blackwell Science Ltd.
DT Journal; General Review
LA English
AB A **review** with 126 refs. Replication of the human immunodeficiency virus (HIV), the causative agent of the acquired immunodeficiency syndrome (AIDS), is under the control of both viral and host factors. Among the latter, the regulatory network of cytokines has been shown to affect virtually every step of the virus life cycle, from cell entry to budding of new progeny virions. Proinflammatory cytokines, such as tumor necrosis factor .alpha., can either trigger or potentiate HIV expression via activation of the cellular transcription factor NF-.kappa.B. Other mols., including interleukin 6 (IL-6) and the interferons, can up-regulate HIV expression by acting predominantly at post-transcriptional and/or post-translational levels. Anti-**inflammatory cytokines**, including **transforming growth factor .beta.**, IL-4 and IL-10, counteract these effects but can also potentiate viral replication under different exptl. conditions. Chemotactic cytokines (chemokines) have recently entered the arena of host factors controlling viral spreading as potent inhibitors competing with the virus for cell-surface 7-transmembrane domain receptors also acting, together with CD4, as entry co-receptors for HIV. The cytokine network is constitutively activated in most HIV-infected individuals, as demonstrated by recent anal. of intracellular signaling mols. such as the Janus kinase/signal transducer and activator of transcription pathway. Finally, cytokines have already shown their potential use as pharmacol. agents able to restore at least some of the compromised immune functions in infected individuals, as exemplified by the potent enhancing effect of IL-2 on the no. of circulating CD4+ T lymphocytes.
RE.CNT 126 THERE ARE 126 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
AN 2000:72099 CAPLUS
DN 132:217203
TI VIP and PACAP38 modulate cytokine and nitric oxide production in peritoneal macrophages and macrophage cell lines
AU Delgado, Mario; Munoz-Elias, Ernesto J.; Martinez, Carmen; Gomariz, Rosa P.; Ganea, Doina
CS Department of Biological Sciences, Rutgers University, Newark, NJ, 07102, USA
SO Annals of the New York Academy of Sciences (**1999**), 897(Neuropeptides), 401-414
CODEN: ANYAA9; ISSN: 0077-8923
PB New York Academy of Sciences

DT Journal; General Review
LA English
AB A **review**, with 39 refs. Macrophages, participants in both innate and specific immunity, have numerous functions, such as phagocytosis, antigen processing and presentation, secretion of both pro- and anti-inflammatory cytokines, prodn. of reactive oxygen and nitrogen intermediates. Following stimulation with microbial products like LPS, macrophages secrete several pro-inflammatory products such as TNF.alpha., IL-12, IL-1, IL-6 and nitric oxide (NO), followed later by the secretion of the anti-**inflammatory cytokines** IL-10 and **TGF.beta.**. Despite their general beneficial role in host defense, the sustained prodn. of pro-inflammatory cytokines and NO can lead to serious pathol. conditions, for example, septic shock, autoimmune diseases, inflammatory bowel disease and respiratory distress syndrome. The ability to control an inflammatory state depends on the local balance between pro- and anti-inflammatory factors. In this respect, a no. of regulatory mols. called "macrophage deactivating factors" have received considerable interest lately. In addn. to anti-**inflammatory cytokines** like IL-10, **TGF.beta.** and IL-13, neuropeptides such as the vasoactive intestinal peptide (VIP) and the pituitary adenylate cyclase activating polypeptide (PACAP) which were previously shown to inhibit the activity of stimulated T cells, and to affect certain macrophage activities, might also function as macrophage deactivating factors. Here the authors report on the effects in vivo and in vitro of VIP and PACAP on the prodn. of TNF.alpha., IL-12, IL-6, IL-10 and NO by LPS-activated peritoneal macrophages and the Raw 264.7 cell line. VIP/PACAP inhibit the prodn. of the pro-inflammatory cytokines TNF.alpha., IL-6 and IL-12, and of nitric oxide, and stimulate the prodn. of the anti-inflammatory cytokine IL-10. In addn., VIP/PACAP exert a protective effect in vivo in a high-endotoxic model for septic shock, presumably by inhibiting the prodn. of endogenous pro-inflammatory cytokines.
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 1999:415083 CAPLUS
DN 131:227247
TI Cytokines in the immunopathogenesis of lupus
AU Handwerger, Barry S.; Luzina, Irina; da Silva, Ludmila; Storrer, Catherine E.; Via, Charles S.
CS Division of Rheumatology, University of Maryland School of Medicine, Baltimore, MD, USA
SO Lupus (**1999**), 321-340. Editor(s): Kammer, Gary M.; Tsokos, George C. Publisher: Humana, Totowa, N. J.
CODEN: 67VNAV
DT Conference; General Review
LA English
AB A **review** with 122 refs. focusing on the roles of type 1 and type 2 cytokines, **inflammatory cytokines**, and **transforming growth factor .beta.** in the pathogenesis of lupus. The reported data strongly suggest that an overprodn. of either type 1 (interferon .gamma.) or type 2 (interleukin-4, -6, and -10) cytokines with B cell-stimulatory activity and/or a relative underprodn. of the assocd. counter-regulatory cytokines leads to polyclonal B cell activation and autoimmunity in lupus. In addn., tissue damage in lupus is assocd. with the local overprodn. of inflammatory cytokines.
RE.CNT 123 THERE ARE 123 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3

AN 1999:487927 CAPLUS
DN 131:331594
TI FK506 nephrotoxicity
AU Finn, William F.
CS Division of Nephrology and Hypertension Department of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, NC, 27599-7155, USA
SO Renal Failure (**1999**), 21(3 & 4), 319-329
CODEN: REFAE8; ISSN: 0886-022X
PB Marcel Dekker, Inc.
DT Journal; General Review
LA English
AB A **review**, with 48 refs. Tacrolimus (FK506) is a potent immunosuppressive agent with significant nephrotoxic properties. FK506 is complexed with an intracellular binding protein FKBP-12. Both the immunosuppressive and nephrotoxic effects may be linked to the inhibitory effect of this complex on calcineurin. The initial phase of FK506 nephrotoxicity is assocd. with a redn. in renal blood flow and glomerular filtration rate. More significant microvascular injury may follow with endothelial damage. Tubular epithelial cell vacuolation, atrophy and microcalcification may be assocd. with the development of irreversible interstitial fibrosis. At times, mesangial cell proliferation adds to the glomerular abnormalities. These effects may be mediated by the inhibitory effect on calcineurin and its role in regulating cellular calcium channels. FK506 stimulates several **inflammatory cytokines**, such as **transforming growth factor-beta**, with potential deleterious effects. Also abnormalities in the renin-angiotensin system, endothelin, renal prostaglandins, adrenergic receptors may all play a role in the nephrotoxic effects.
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
AN 1999:137039 CAPLUS
DN 130:279938
TI The role and regulation of phospholipase A2 in periodontitis
AU Shinohara, Hiroyuki; Nagata, Toshihiko
CS Sch. Dent., Univ. Tokushima, Tokushima, 770-8504, Japan
SO Shikoku Shigakkai Zasshi (**1999**), 11(2), 201-216
CODEN: SSZAED; ISSN: 0914-6091
PB Shikoku Shigakkai
DT Journal; General Review
LA Japanese
AB A **review** with 92 refs. Phospholipase A2 (PLA2) hydrolyzes the sn-2 ester bond of phospholipids. PLA2 is a key enzyme in the prodn. of potent inflammatory mediators, including prostaglandins (PG), leukotrienes and platelet activating factor. Although more than 10 isoenzymes of PLA2 were recently identified, group II secretory PLA2 (II-PLA2) has been clarified to be implicated in the inflammatory process. II-PLA2 activity in human gingival crevicular fluid (GCF) and gingival tissue from patients with periodontitis reflected periodontal disease conditions. In vitro study using gingival fibroblasts demonstrated that II-PLA2 was induced and secreted by the inflammatory cytokines, such as interleukin-1.beta. (IL-1.beta.) and tumor necrosis factor-.alpha. (TNF .alpha.), and bacterial lipopolysaccharide (LPS), but it was inhibited by the anti-**inflammatory cytokine transforming growth factor .beta.**. The bone resorption factors detected in GCF are IL-1.beta., TNF.alpha., LPS and PGE2, which have been reported to serve as markers of periodontal tissue destruction. These factors were assocd. with PLA2-related events. Moreover, the increase of gingival PLA2 activity preceded alveolar bone resorption in exptl. periodontitis. Taken together the PLA2 activity in GCF of patients

with periodontitis can be used as a diagnostic marker for periodontal disease activity.

L9 ANSWER 8 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 5
AN 2000:62559 BIOSIS
DN PREV200000062559
TI AP-1: One switch for many signals.
AU Wisdom, Ron (1)
CS (1) Department of Biochemistry, Vanderbilt University, Nashville, TN USA
SO Experimental Cell Research, (**Nov. 25, 1999**) Vol. 253, No. 1, pp. 180-185.
ISSN: 0014-4827.
DT General Review
LA English
SL English
AB The transcription factor AP-1 is activated in response to an incredible array of stimuli, including mitogenic growth factors, **inflammatory cytokines**, growth factors of the **TGF-beta** family, UV and ionizing irradiation, cellular stress, antigen binding, and neoplastic transformation. In this **review**, I discuss genetic evidence that supports a role for AP-1 in the cellular response to some of these stimuli and describe biochemical properties that might explain the ability of this transcription factor to activate different sets of genes in response to different stimuli.

L9 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 2000:263924 CAPLUS
DN 133:38274
TI Possible role of microglial prostanoids and free radicals in neuroprotection and neurodegeneration
AU Minghetti, Luisa; Polazzi, Elisabetta; Nicolini, Alessia; Greco, Anita; Levi, Giulio
CS Neurobiology Section Laboratory of Pathophysiology Istituto Superiore di Sanita, Rome, 00161, Italy
SO Advances in Experimental Medicine and Biology (**1999**), 468(Functional Roles of Glial Cells in Health and Disease), 109-119
CODEN: AEMBAP; ISSN: 0065-2598
PB Kluwer Academic/Plenum Publishers
DT Journal; General Review
LA English
AB A **review**, with 76 refs. The following topics were discussed: microglial prostanoids and neurodegeneration; NO and prostanoid synthesis in microglial cultures from neonatal and adult rat brain; prostanoid and NO reciprocal regulation; and effects of cAMP elevating agents, pro-**inflammatory cytokines** (INF.gamma.), and anti-inflammatory and immunosuppressive agents (**TGF**-.**beta**.1, IL-10, and lipocortin-1) on microglial prostanoid and NO synthesis.
RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L9 ANSWER 10 OF 25 COPYRIGHT 2002 Gale Group

AN 1998:53221 NLDB
TI AUTOIMMUNE STARTING PHASE III OF RHEUMATOID ARTHRITIS DRUG
SO BIOWORLD Today, (**25 Feb 1998**) Vol. 9, No. 36.
PB American Health Consultants Inc.
DT Newsletter
LA English
WC 775

L9 ANSWER 11 OF 25 COPYRIGHT 2002 Gale Group

AN 1998:160159 NLDB
TI Journal News . . . June 29, 1998 **Reviews** and Information From Periodicals and Journals Worldwide . . . Compiled by Alan D. Henderson
SO Gene Therapy Weekly, (**29 Jun 1998**) .
ISSN: 1078-2842.
PB Charles W Henderson
DT Newsletter
LA English
WC 284

L9 ANSWER 12 OF 25 USPATFULL
AN 1998:115616 USPATFULL
TI TNF-.alpha. ribozymes
IN Sullivan, Sean, Alameda, CA, United States
Draper, Kenneth, Boulder, CO, United States
Kisich, Kevin, Lafayette, CO, United States
Stinchcomb, Dan T., Boulder, CO, United States
McSwiggen, James, Boulder, CO, United States
PA Ribozyme Pharmaceuticals, Inc., Boulder, CO, United States (U.S. corporation)
PI US 5811300 19980922 <--
AI US 1994-311486 19940923 (8)
DCD 20150504
RLI Continuation-in-part of Ser. No. US 1992-989849, filed on 7 Dec 1992, now abandoned And Ser. No. US 1993-8895, filed on 19 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: LeGuyader, John L.
LREP Lyon & Lyon LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1,9,10
DRWN 15 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 7400
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Enzymatic RNA molecules which cleave TNF-.alpha. mRNA.

L9 ANSWER 13 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6
AN 1999:108743 CAPLUS
DN 130:323990
TI Cytokines as intrinsic and exogenous regulators of pathogenesis in experimental autoimmune encephalomyelitis
AU Begolka, W. Smith; Miller, S. D.
CS Department of Microbiology-Immunology and Interdepartmental Immunobiology Center, Northwestern University Medical School, Chicago, IL, USA
SO Research in Immunology (**1998**), 149(9), 771-781
CODEN: RIMME5; ISSN: 0923-2494
PB Editions Scientifiques et Medicales Elsevier
DT Journal; General Review
LA English
AB A **review** with approx. 100 refs. focusing on the current understanding of the often contradictory results pertaining to the regulatory roles for both pro- and anti-inflammatory cytokines as immune mediators during the course of exptl. autoimmune encephalomyelitis, a prototypic animal model of multiple sclerosis. Pro-inflammatory cytokines discussed are: interleukin-12, interferon .gamma., and tumor necrosis factor .alpha./lymphotoxin .alpha. (tumor necrosis factor .beta.). Anti-**inflammatory cytokines** discussed are: interleukin-10, interleukin-4, and **transforming growth factor .beta.**.

RE.CNT 102 THERE ARE 102 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 14 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 7
AN 1998:584417 CAPLUS
DN 129:342426
TI A gene therapy approach to treat demyelinating diseases using non-replicative herpetic vectors engineered to produce cytokines
AU Martino, G.; Furlan, R.; Galbiati, F.; Poliani, P. L.; Bergami, A.; Grimaldi, L. M. E.; Adorini, L.; Comi, G.
CS Exp. Neuroimmunotherapy Unit - DIBIT, San Raffaele Scientific Institute, Milan, 20132, Italy
SO Multiple Sclerosis (**1998**), 4(3), 222-227
CODEN: MUSCFZ; ISSN: 1352-4585
PB Stockton Press
DT Journal; General Review
LA English
AB A **review** and discussion with 37 refs. A successful gene therapy approach in organ-specific autoimmune diseases, such as multiple sclerosis (MS), encompasses the inhibition of the autoreactive T cells or the modification of the target organ cells by the introduction of exogenous "protective" genes. In MS, an autoimmune disease of the central nervous system (CNS), the inciting autoantigen is still unknown and therefore the isolation of autoreactive T cells may only be inferential. At present, gene therapy approaches in MS should therefore aim to the modification of the target organ. Possible candidate genes to be transferred within the CNS of MS patients are those coding for anti-**inflammatory cytokines** (i.e. interleukin-4, interleukin-10, **transforming growth factor .beta.**) which have been shown to ameliorate demyelinating diseases at least in exptl. models. However, a limiting factor for this therapy is the difficulty to reach the CNS. A gene therapy approach using viral vectors able to infect post-mitotic cells, such as those present within the CNS, without inducing toxic reactions, may overcome this limitation. We propose to use non-replicative herpetic vectors, which represent a viable gene-transfer alternative to the classical retroviral and adenoviral vectors. Key advantages are their size, able to accommodate multiple foreign genes, and their ability to infect post-mitotic cells such as those present within the CNS. We first transferred a gene coding for interleukin-4 within the CNS of mice undergoing exptl. allergic encephalomyelitis, an animal model for MS, using non-replicative Herpes Simplex Virus type 1-derived vectors. We found that this approach ameliorates the disease course and delays the disease onset. The establishment of this technique to deliver anti-inflammatory cytokines within the CNS using herpetic vectors should clarify the role of individual cytokines in the demyelinating process and allow assessment of whether gene therapy using herpetic vectors is a feasible and safe approach to treat human demyelinating disorders.

L9 ANSWER 15 OF 25 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1998133735 EMBASE
TI [Cytokines and peripheral neuropathies].
CYTOKINES ET NEUROPATHIES PERIPHERIQUES.
AU Creange A.; Lefaucheur J.-P.; Authier F.-J.; Gherardi R.-K.
CS A. Creange, Laboratoire GERMEN, Faculte de Medecine de Creteil, 8, rue du General Sarrail, F-94010 Creteil Cedex, France. creange@univ-paris12.fr
SO Revue Neurologique, (1998) 154/3 (208-216).
Refs: 86
ISSN: 0035-3787 CODEN: RENEAM
CY France
DT Journal; General Review
FS 008 Neurology and Neurosurgery

026 Immunology, Serology and Transplantation
LA French
SL English; French
AB Cytokines are polypeptides produced by various cells, with key-roles in regulation of immune response, inflammation and hematopoiesis. Cytokine-producing cells in peripheral nerve include resident and recruited macrophages, lymphocytes, and likely mastocytes, Schwann cells, and probably neurons. Cytokines are instrumental in pathogenesis of peripheral neuropathies during nerve lesions and tissue repair. Tumor necrosis factor- alpha (TNF-.alpha.) injection into nerve induces Wallerian degeneration. In contrast, interleukin-1 (IL-1) promotes detersion by scavenger macrophages, and increased synthesis of neurotrophic factors (nerve growth factor - NGF - and leukemia inhibitory factor -LIF). Neurotrophic cytokines IL-6, LIF and transforming growth factor-beta 1 (TGF-.beta.1) are overexpressed in nerve after experimental axotomy and promote axonal growth until axon/Schwann cell contact. In the course of inflammatory demyelinating neuropathies, proinflammatory cytokines induce vascular permeability and breakdown of blood nerve barrier (TNF-.alpha., vascular endothelial growth factor/vascular permeability factor - VEGF/VPF), favor leukocyte transmigration into nerve, induce activation and proliferation of lymphocytes (IL-1, IL-2) and macrophages (gamma-interferon - IFN-.gamma.), and have a direct myelinotoxic activity (TNF-.alpha. and TNF-.beta.). In addition, the inflammatory process is likely favored by downregulation of the anti-**inflammatory cytokine TGF-.beta.**1.

L9 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 8
AN 1998:164066 CAPLUS
DN 128:179090
TI TGF-.beta. in renal allograft rejection
AU Cohen, Arthur H.; Nast, Cynthia C.
CS Department Pathology, Cedars-Sinai Medical Center, Los Angeles, CA, 90048, USA
SO Mineral and Electrolyte Metabolism (**1998**), 24(2-3), 197-201
CODEN: MELMDI; ISSN: 0378-0392
PB S. Karger AG
DT Journal; General Review
LA English
AB A **review** with 26 refs. The role of TGF-.beta. in pathol. processes in the transplanted kidney is beginning to be investigated both in animal models and in humans. In both settings in acute cell-mediated rejection, TGF-.beta., receptor, and message have all been documented to be elevated in the tubulointerstitium, likely a reflection of TGF-.beta.'s role in recruiting leukocytes to areas of injury and downregulation of the inflammatory response. In chronic rejection, expression of TGF-.beta., message, and induced proteins is elevated, esp. in cortex. **TGF -.beta.** mRNA, unlike other **inflammatory cytokine** mRNAs, correlated very well with interstitial fibrosis, a hallmark of chronic rejection. Thus, a relationship between renal scarring and TGF-.beta. has been documented by most studies of transplant kidneys. Addnl., this growth factor also appears to have a role in the renal fibrosis assocd. with cyclosporine administration and perhaps in augmenting this drug's immunosuppressive effects.

L9 ANSWER 17 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 9
AN 1999:474817 CAPLUS
DN 131:298906
TI Effects of calorie restriction and .omega.-3 dietary fat on aging in short- and long-lived rodents
AU Troyer, Dean A.; Venkatraman, Jaya T.; Fernandes, Gabriel
CS Department of Pathology, The University of Texas Health Science Center at San Antonio, San Antonio, TX, 7828, USA

SO Age (Media, Pennsylvania) (**1998**), 21(4), 175-182
CODEN: AGEEDB; ISSN: 0161-9152
PB American Aging Association
DT Journal; General Review
LA English
AB A **review** with 74 refs. Aging is accompanied by a steady increase in the incidence of spontaneous tumors and a decline in immune function. Calorie restriction (CR) or supplementation with .omega.-3 fats prolongs life span, suppresses tumorigenesis, and ameliorates immune function in a variety of exptl. models. We suggest that decreased oxidant stress and upregulation of apoptosis mediate the effects of calorie restriction on immunity and longevity. CR prolongs life span in several animal models and our studies have examd. the effects of CR on the immune system and on tumorigenesis. CR maintains naive T cells, prevents the rise in "double-neg." T cells, maintains lymphocyte responsiveness to mitogens, and preserves Dexamethasone induced apoptosis in spleen cells of MRL/lpr mice. CR also modulates the expression of inflammatory mediators and cytokines. CR decreases the Sjogren's syndrome-like chronic inflammation of salivary glands of B/W animals while increasing expression of the immunosuppressive cytokine **TGF.beta**.1 and decreasing expression of the pro-**inflammatory cytokines** IL-6 and TNF.alpha.. The autoimmune disease in the B/W mouse also affects the kidneys, and we find that renal expression of platelet derived growth factor-A, (PDGF-A) and thrombin receptor are decreased in CR animals. Similarly, CR decreases the expression and localization of plasminogen activator inhibitor type 1 in glomeruli of B/W animals. CR also modulates expression and function of androgen receptors and the binding of insulin to liver nuclei. Finally, CR suppresses the development of breast tumors in the Ras *oncomouse*. These effects of calorie restriction are paralleled in short-lived B/W animals fed diets supplemented with .omega.-3 fatty acids. Omega-3 fatty acids induce the expression of hepatic antioxidant enzymes, and enhance apoptosis in lymphocytes of B/W animals.
RE.CNT 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 10
AN 1998:303020 CAPLUS
DN 129:49062
TI Termination of acute-phase response: role of some cytokines and anti-inflammatory drugs
AU Koj, Aleksander
CS Department of Metabolic Regulations, Institute of Molecular Biology, Jagiellonian University, Krakow, 31-120, Pol.
SO General Pharmacology (**1998**), 31(1), 9-18
CODEN: GEPHDP; ISSN: 0306-3623
PB Elsevier Science Inc.
DT Journal; General Review
LA English
AB A **review** with many refs. on the approach in effective termination of acute-phase response by combined use of anti-inflammatory cytokines and specific drugs. The acute-phase response is triggered by changes in intracellular mediators that activate stress-sensitive kinases and transcription factors controlling the synthesis of proinflammatory cytokines such as TNF-.alpha., IL-1, IL-8 or IFN-.gamma.. Natural extinguishing of acute-phase response occurs due to short half-lives of inflammatory mediators and prodn. of anti-**inflammatory cytokines** such as IL-10, IL-4, IL-13, **TGF**-.**beta**. and some others. Excess proinflammatory cytokines are removed by sol. cytokine receptors and receptor antagonists. Synthesis of proinflammatory mediators and cytokines can be blocked by glucocorticoids, some nonsteroidal anti-inflammatory drugs suppressing cyclooxygenase and by specific inhibitors of cytokine induction.

L9 ANSWER 19 OF 25 USPATFULL
AN 97:117893 USPATFULL
TI Detecting genetic predisposition for osteoporosis
IN Duff, Gordon W., 18 Ashgate Road, Sheffield, S10 3BZ, S Yorks, England
Russell, Graham, Ronksley Farm Hollow Meadows, Sheffield, South Yorks S6 6GH, England
Eastell, Richard, 289 Ringinglow Road, Sheffield, S11 7PZ, England
PI US 5698399 19971216 <--
AI US 1996-628282 19960405 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Horlick, Kenneth R.
LREP Jenkens & Gilchrist
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 668
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods of predicting the risk of osteoporosis. Specifically, the methods comprise isolating genomic DNA from an individual and determining an allelic pattern for IL-1 receptor antagonist (IL-1ra) in the genomic DNA. The identification of at least one copy of allele 2 indicates increased susceptibility to osteoporosis.

L9 ANSWER 20 OF 25 USPATFULL
AN 97:63997 USPATFULL
TI Methods of modulating **inflammatory cytokines** in the CNS using **TGF-.beta.**
IN Carlino, Joseph A., San Leandro, CA, United States
Benveniste, Etty N., Birmingham, AL, United States
PA Celtrix Pharmaceuticals, Inc., Santa Clara, CA, United States (U.S. corporation)
PI US 5650396 19970722 <--
AI US 1994-213001 19940315 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Killos, Paul J.
LREP Morrison & Foerster
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1134
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for modulating the expression of inflammatory cytokines in the central nervous system comprising administering an effective amount of **TGF-.beta.** are disclosed. The methods include suppressing pro-**inflammatory cytokines** in the central nervous system by administering an effective amount of **TGF-.beta.** and inducing anti-**inflammatory cytokines** in the central nervous system by administering an effective amount of TGF-.beta..

L9 ANSWER 21 OF 25 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 11
AN 1996:234370 CAPLUS
DN 124:286210
TI Role of cytokines in rheumatoid arthritis
AU Feldmann, Marc; Brennan, Fionula M.; Maini, Ravinder N.
CS Mathilda and Terence Kennedy Institute Rheumatology, London, W6 8LW, UK
SO Annual Review of Immunology (**1996**), 14, 397-440
CODEN: ARIMDU; ISSN: 0732-0582
PB Annual Reviews

DT Journal; General Review
LA English
AB A **review**, with 226 refs. Anal. of cytokine mRNA and protein in rheumatoid arthritis tissue revealed that many proinflammatory cytokines such as TNF.alpha., IL-1, IL-6, GM-CSF, and chemokines such as IL-8 are abundant in all patients regardless of therapy. This is compensated to some degree by the increased prodn. of anti-**inflammatory cytokines** such as IL-10 and **TGF.beta.** and cytokine inhibitors such as IL-1ra and sol. TNF-R. However, this upregulation in homeostatic regulatory mechanisms is not sufficient as these are unable to neutralize all the TNF.alpha. and IL-1 produced. In rheumatoid joint cell cultures that spontaneously produce IL-1, TNF.alpha. was the major dominant regulator of IL-1. Subsequently, other proinflammatory cytokines were also inhibited if TNF.alpha. was neutralized, leading to the new concept that the proinflammatory cytokines were linked in a network with TNF.alpha. at its apex. This led to the hypothesis that TNF.alpha. was of major importance in rheumatoid arthritis and was a therapeutic target. This hypothesis has been successfully tested in animal models, of, for example, collagen-induced arthritis, and these studies have provided the rationale for clin. trials of anti-TNF.alpha. therapy in patients with long-standing rheumatoid arthritis. Several clin. trials using a chimeric anti-TNF.alpha. antibody have shown marked clin. benefit, verifying the hypothesis that TNF.alpha. is of major importance in rheumatoid arthritis. Retreatment studies have also shown benefit in repeated relapses, indicating that the disease remains TNF.alpha. dependent. Overall these studies demonstrate that anal. of cytokine expression and regulation may yield effective therapeutic targets in inflammatory disease.

L9 ANSWER 22 OF 25 CANCERLIT DUPLICATE 12
AN 95382492 CANCERLIT
DN 95382492 PubMed ID: 7653937
TI Cytokines in Sjogren's syndrome.
AU Skopouli F N; Moutsopoulos H M
CS Dept of Internal Medicine, Medical School, University of Ioannina, Greece.
SO ANNALES DE MEDECINE INTERNE, **(1995)** 146 (4) 219-22. Ref: 30
Journal code: 0171744. ISSN: 0003-410X.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 95382492
EM 199509
ED Entered STN: 19951108
Last Updated on STN: 19951108
AB This **review** discusses the respective role in Sjogren's syndrome of pro-inflammatory cytokines, such as interleukin (IL)-1 beta and tumour-necrosis factor-alpha, and anti-**inflammatory cytokines**, such as **transforming growth factor-beta**, interferon-alpha and IL-10. The former products are secreted by lymphocytes as well as epithelial cells. Th 1 cell cytokines predominate within the focal infiltrates. Increased levels of circulating IL2 receptors correlate to the progression of the ongoing disease.

L9 ANSWER 23 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 1994:646384 CAPLUS
DN 121:246384
TI Transforming growth factor-.beta. (TGF.beta.) in vascular endothelial cells

AU Hirai, Reiko
CS Tokyo Metrop. Inst. Med. Sci., Tokyo, 113, Japan
SO Igaku no Ayumi (**1994**), 170(5), 420-4
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A **review**, with 11 refs., on the structures of active and latent forms of TGF.beta., and its function elucidated by TGF.beta. knock out mice, which is useful as a disease model for autoimmune diseases and graft-vs.-host disease (GVH). Two TGF.beta. receptors have been elucidated, and some other proteins exhibit affinity to TGF.beta.. TGF.beta. exhibits complex stimulation and suppression effects on endothelial cells depending on the cells and TGF.beta. subtypes, and induces ductal formation by acting on endothelial cells of microvessels. **TGF.beta.** suppresses prodn. of **inflammatory cytokines**, and adhesion of neutrophils. Finally, TGF.beta. induces endothelin expression.

L9 ANSWER 24 OF 25 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE
AN 1994:24205841 BIOTECHNO
TI Cells, matrix, growth factors, and the surgeon: The biology of scarless fetal wound repair
AU Adzick N.S.; Lorenz H.P.
CS Fetal Treatment Center, University of California, 3rd and Parnassus Avenues,San Francisco, CA 94143-0570, United States.
SO Annals of Surgery, (**1994**), 220/1 (10-18)
CODEN: ANSUA5 ISSN: 0003-4932
DT Journal; General Review
CY United States
LA English
SL English
AB Objective: This **review** updates the surgeon about the cellular, matrix, and growth factor components of scarless fetal wound repair. Summary Background Data: Fetal skin wound healing is characterized by the absence of scar tissue formation. This unique repair process is not dependent on the sterile, aqueous intrauterine environment. The differences between fetal and adult skin wound healing appear to reflect processes intrinsic to fetal tissue, such as the unique fetal fibroblasts, a more rapid and ordered deposition and turnover of tissue components, and, particularly, a markedly reduced inflammatory infiltrate and cytokine profile. Scarless fetal wounds are relatively deficient in the **inflammatory cytokine, transforming growth factor .beta.** (**TGF-.beta.**). In contrast, the fibrosis characteristic of adult wound repair may be associated with TGF-.beta. excess. Recent experimental studies suggest that specific anti-TGF-.beta. therapeutic strategies can ameliorate scar formation in adult wound repair and fibrotic diseases. Inhibitors of TGF-.beta. may be important future drugs to control scar. Conclusions: Based on the scarless fetal wound repair model, a number of ways in which the matrix and cellular response of the healing adult wound might be manipulated to reduce scarring are reviewed.

L9 ANSWER 25 OF 25 CANCERLIT DUPLICATE 14
AN 92199024 CANCERLIT
DN 92199024 PubMed ID: 1550874
TI Relationship of TNF to interleukins.
AU Neta R; Sayers T J; Oppenheim J J
CS Armed Forces Radiobiology Research Institute, Bethesda, Maryland.
NC NO1-CO-74102 (NCI)
SO IMMUNOLOGY SERIES, **(1992)** 56 499-566. Ref: 364
Journal code: 0404721. ISSN: 0092-6019.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 92199024
EM 199204
ED Entered STN: 19941107
Last Updated on STN: 19941107
AB It is evident from this **review** that TNF exhibits complex interactions with other cytokines at the level of production and in its effects. Studies designed to determine the role of TNF in the animal models or cell culture system using pure recombinant molecules have revealed that TNF never operates by itself, but instead operates within a network of cytokines. First, the multitude of exogenous as well as endogenous signals, which induce TNF production, concomitantly also stimulate the production of a battery of other **inflammatory cytokines**: IL-1, IL-6, IL-8, multiple CSFs, IFN, and **TGF -beta**. Moreover, TNF itself stimulates the production of most of these cytokines. Thus even when pure recombinant TNF is used, it readily generates the production of other interactive cytokines. This apparent redundancy in the production of cytokines with overlapping effects presumably has protective advantage for the host. Furthermore, interaction of these cytokines is more economical and amplifies the responses to subtoxic doses of potentially harmful cytokines. Cytokine interaction may lead to either synergistic (as for many TNF-IL-1 interactions) or antagonistic effects (TNF and TGF-beta, for example). These may depend on (1) the modulation of receptor expression of one cytokine by another (IFN-gamma-enhancing receptor expression for TNF, and TGF-beta down-regulation of IL-1 receptors), (2) stabilization of the cytokine message by one another (induction of IL-6 by TNF or IL-1), (3) interactions at the level of signal transduction, (4) gene expression, or (5) at the posttranslational level. Thus the receptor repertoire, which is a function of the cell type and stage of development, actually determines the net effects of a particular combination of interactive cytokines. Clearly, the mechanisms of these interactions will need to be elucidated to better understand their biological function and to permit cytokines to be used clinically to the advantage of the host.

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=> s l5 (10A) (inflammatory cytokine)
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59 FILES SEARCHED...
L10 0 L5 (10A) (INFLAMMATORY CYTOKINE)

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CR   2002-339939 [37]; 2002-339941 [37]
DNC C2002-146748
TI   New pyridine derivatives are **IkappaB kinase inhibitors** used for treating e.g. asthma, ischemia, sepsis, psoriasis and gout.
DC   B02 B03
IN   FUCHIKAMI, K; KADONO, H; KOMURA, H; KORIYAMA, Y; LOWINGER, T B; MASUDA, T; MURATA, T; SAKAKIBARA, S; SATO, H; SHIMADA, M; SHINTANI, T; UMEDA, M; YOSHINO, T; ZIEGELBAUER, K B
PA   (FARB) BAYER AG; (LOWI-I) LOWINGER T B
CYC 97
PI   WO 2002024679 A1 20020328 (200255)* EN 280p
        RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
            NL OA PT SD SE SL SZ TR TZ UG ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
            KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO
            RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
     AU 2001089873 A 20020402 (200255)
ADT WO 2002024679 A1 WO 2001-EP10405 20010910; AU 2001089873 A AU 2001-89873 20010910
FDT AU 2001089873 A Based on WO 200224679
PRAI JP 2000-289173   20000922
AB   WO 200224679 A UPAB: 20020829
     NOVELTY - Pyridine derivatives (I) are new.
          DETAILED DESCRIPTION - Pyridine derivatives of formula (I) and their salts are new.
          R1 = 3-hydroxypyridin-2-yl, 3-hydroxythiophen-2-yl or R11 substituted 2-hydroxyphenyl;
          R11 = H, halo, OH, 1-12C alkoxy, NO2, amino, 1-6C alkylsulfonylamino, 1-6C alkoxycarbonyl, 1-6C alkylamino, di(1-6C alkyl)amino, 1-6C alkanoylamino, phenyl 1-6C alkylamino, phenylsulfonylamino or O-(CH2)n-R111;
          n = 0-6;
          R111 = 2-6C alkenyl, benzoyl, diphenylmethyl, di(1-6C alkyl)amino, 1-6C alkanoyl, 1-6C alkoxycarbonyl or 3-10 membered ring optionally having 1-3 S, O or N heteroatoms (optionally substituted by 1-6C alkyl, mono or di halo, 1-6C haloalkyl, NO2, CN, 1-6C alkoxycarbonyl, phenyl, OH, amino, 1-6C alkylamino, di(1-6C alkyl)amino, 1-6C alkanoylamino, 1-6C alkoxy or carbamoyl;

R2 = H or halo;
R3 = H, 1,2,3,6-tetrahydropyridine, -CR31R32R33 or NR34R35;
R31 = H or 1-6C alkyl;
R32 = H, alpha -aminobenzyl, 1-6C alkyl (optionally substituted by 1 or 2 hydroxy, amino, optionally substituted phenyl, halo substituted phenyl or 1-6C alkoxy substituted phenyl), or 5-8 membered saturated ring optionally having 1-3 S, O or N heteroatoms (optionally substituted by 1-6C alkyl);
R33 = H, amino, 1-6C alkoxy carbonylamino, 2-6C alkenyloxycarbonylamino, piperidino-1-6C alkylcarbonylamino or piperidinyl-1-6C alkylcarbonylamino, or
CR32R33 = 5-8 membered saturated ring optionally having 1-3 N, O or S heteroatoms (optionally substituted by phenyl-1-6C alkyl, 1-6C alkoxy substituted phenyl 1-6C alkyl, 1-6C alkyl, amino, CN, carbamoyl, carboxy, 1-6C alkylamino, di(1-6C alkyl)amino, benzylamino, 1-6C alkylsulfonyl, piperidino 1-6C alkyl carbonyl or optionally fused by benzene);
R34 = H or 1-6C alkyl;
R35 = H, 5-8 membered saturated ring optionally having 1-3 N, O or S heteroatoms, or (CH2)m-NR351R352, or
NR34R35 = 5-8 membered saturated heterocyclyl optionally having NH, S or O atoms other than the adjacent N atom and substituted by carbamoyl, amino or 1-6C alkyl);
m = 1-6;
R351 = H or 1-6C alkyl;
R352 = H, 1-6C alkyl, 1-6C alkanoyl, 1-6C alkyl substituted phenyl, benzoyl, 1-6C alkanoyl, phenylaminocarbonyl or phenylsulfonyl;
R4 = hydroxycarbonyl, 1-6C alkanoyl, carbamoyl, CN, NO2, carboxyl, 1-6C alkoxycarbonyl, 1-6C alkylcarbamoyl, 1-6C alkylamino, 5-10 membered heteroaryl(hydroxy)methyl, 5-10 membered heteroaryl 1-6C alkyl or methyl substituted by hydroxy, or 5-7 membered saturated cyclic ring or 1-6C alkyl (optionally substituted by OH, 1-6C alkoxy, 1-6C alkylsulfonylamino, 1-6C alkylcarbonyl amino, 5-10C aryl, 5-10C arylsulfonyl, 5-10C arylsulfanyl, 5-10C aryloxy, imidazolyl, or dioxo substituted pyrrolidino-oxy), -(CH2)pNHCOR41 or -(CH2)pNHC(=S)R41, or
CR3CR4 = 4-10 membered mono- or bi-cycloalkyl (optionally substituted by benzyl, =NH or =O and optionally interpreted by NH);
p = 1-6;
R41 = 1-6C alkoxy, amino, phenylamino, 1-6C alkyl, 1-6C alkylamino, di(1-6C alkyl)amino or 3 - 10 cycloalkylamino;
R5 = NR51R52, or
R4 + R5 = R40-CO-NH-, R40-SO2-NH-, R40-C(=S)-NH- or R40-CH2-NH-;
R51 = H or 1-6C alkyl;
R52 = H, 1-6C alkyl, phenyl, benzyl or 1-6C alkanoyl, or
NR51R52 = optionally saturated 5-6 membered ring optionally containing NH or O other than adjacent N;
R40 = -CHR401-O-, -CH2N-R401, -CO-NR401, -CH2CHR401, -CH=CR401, -CR41=N-NH- or -CR42=N-C=N-;
R401 = 1-6C alkanoyl, 1-6C alkyl, phenyl, 1-6C alkylsulfonyl, 3-8C cycloalkylaminocarbonyl, H, halo, NO2, amino, CN, benzoylamino, phenylsulfonyl, carbamoyl, hydroxycarbonyl, 1-6C alkoxycarbonyl, 1-12C alkylaminocarbonyl, halo substituted 1-6C alkylaminocarbonyl, 1-6C alkanoylamino, 1-6C alkylamino, di(1-6C alkyl)aminocarbonyl, di(1-6C alkyl)1-6C aminoalkylaminocarbonyl, hydroindenylaminocarbonyl, diphenylamethylaminocarbonyl, pyrrolidinocarbonyl, 1-6C alkoxy 1-6C alkyl aminocarbonyl, morpholinocarbonyl, piperazinocarbonyl, phenyl1-6C alkylaminocarbonyl, hydroxycarbonyl 1-6C alkylaminocarbonyl, 3-8C cycloalkylaminocarbonyl, 3-8C cycloalkyl 1-6C alkylaminocarbonyl, hydroxy 1-6C alkylaminocarbonyl, carboxyethylaminocarbonyl or 1-6C alkylsulfonylaminocarbonyl, and
R41 = H, amino or 1-6C alkanoylamino, and
R42 = H or amino.
ACTIVITY - Antiinflammatory; Immunosuppressive; Cytostatic; Antiasthmatic; Antiallergic; Dermatological; Antirheumatic; Antipsoriatic; Antibacterial; Antigout; Vasotropic.

MECHANISM OF ACTION - IkappaB kinase beta (IKK- beta) inhibitor; Nuclear factor kappa B (NF-kB) inhibitor.

In a kinase assay of IKK- beta using recombinant IKK- beta (0.6 mu g/ml) and bio-GST-IkappaB alpha (0.2 mu M) diluted in 2 multiply kinase buffer beta (25 mu l), 2-amino-6-(2-(benzyloxy)-6-hydroxyphenyl)-4-(3-piperidinyl)nicotinonitrile (Ia) exhibited an in vitro IC50 value of less than 0.5 mu M.

USE - Used for treating asthma, allergic rhinitis, atopic dermatitis, hives, conjunctivitis, vernal catarrh, chronic arthrorheumatism, systemic lupus erythematosus, psoriasis, diabrotic colitis, systemic inflammatory response syndrome (SIRS), sepsis, polymyositis, dermatomyositis , polyaritis nodosa, mixed connective tissue disease, Sjoegren's syndrome, gout and ischemia.

ADVANTAGE - (I) Have effective antiinflammatory action based on a specific and selective inhibitory activity to NF-kB.

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L2 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1

AN 2002:393761 BIOSIS

DN PREV200200393761

TI Cytokines modulate telomerase activity in a human multiple myeloma cell line.

AU Akiyama, Masaharu; Hideshima, Teru; Hayashi, Toshiaki; Tai, Yu-Tzu; Mitsiades, Constantine S.; Mitsiades, Nicholas; Chauhan, Dharminder; Richardson, Paul; Munshi, Nikhil C.; Anderson, Kenneth C. (1)

CS (1) Dana-Farber Cancer Institute, 44 Binney Street, Boston, MA, 02115: Kenneth_anderson@dfci.harvard.edu USA

SO Cancer Research, (July 1, 2002) Vol. 62, No. 13, pp. 3876-3882. http://cancerres.aacrjournals.org/. print. ISSN: 0008-5472.

DT Article

LA English

AB Telomerase is a ribonucleoprotein DNA polymerase that elongates the telomeres of chromosomes to compensate for losses that occur with each round of DNA replication and maintain chromosomal stability. Interleukin 6 (IL-6) and insulin-like growth factor 1 (IGF-1) are proliferative and survival factors for human multiple myeloma (MM) cells. To date, however, the effects of IGF-1 and IL-6 on telomerase activity and associated sequelae in MM cells have not been characterized. In this study, we evaluated the effects of IGF-1 and IL-6 on telomerase activity in MM cell lines (MM.1S, U266, and RPMI 8226), as well as patient MM cells. We show that these cytokines up-regulate telomerase activity without alteration of human telomerase reverse transcriptase (hTERT) protein expression. We also demonstrate that increased telomerase activity triggered by these cytokines is mediated by phosphatidylinositol 3'-kinase (PI3k)/Akt/nuclear factor kappaB (NFkappaB) signaling. We confirm involvement of PI3k/Akt/NFkappaB signaling because the PI3k inhibitors wortmannin and LY294002 or the inhibitor of NFkappaB (**IkappaB**) **kinase inhibitor** PS-1145 block constitutive and cytokine-induced up-regulation of telomerase activity. Furthermore, we show that dexamethasone (Dex) reduces telomerase activity through the inhibition of hTERT expression before the induction of apoptosis. Importantly, IGF-1 and IL-6 abrogate Dex-induced down-regulation of telomerase activity and apoptosis. The protective effect of those cytokines against Dex-induced down-regulation of telomerase activity is blocked by both wortmannin and PS-1145, whereas the protection against Dex-induced apoptosis is blocked by wortmannin but not PS-1145. Therefore, our results demonstrate that telomerase activity is related not only to transcriptional regulation of hTERT by NFkappaB but also to post-transcriptional regulation because of phosphorylation of hTERT by Akt kinase. These studies therefore demonstrate that telomerase activity is associated with cell growth, survival, and drug resistance in MM cells.

L2 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2

AN 2002:414661 BIOSIS
DN PREV200200414661
TI The **IkappaB kinase inhibitor** sulfasalazine impairs long-term memory in the crab Chasmagnathus.
AU Merlo, E.; Freudenthal, R.; Romano, A. (1)
CS (1) Laboratorio de Neurobiologia de la Memoria, Facultad de Ciencias Exactas y Naturales, Departamento de Ciencias Biologicas, Universidad de Buenos Aires, Pabellon II, 1428, Buenos Aires: aromano@bg.fcen.uba.ar Argentina
SO Neuroscience, (12 June, 2002) Vol. 112, No. 1, pp. 161-172. http://www.elsevier.com/locate/neuroscience. print. ISSN: 0306-4522.
DT Article
LA English
AB Evidence for the participation of Rel/NF-kappaB transcription factors in long-term memory has recently been reported in the context-signal learning paradigm of the crab Chasmagnathus, in which a high correlation between long-term memory formation and NF-kappaB activation was observed. Two components of the NF-kappaB pathway in the crab brain have now been identified by cross-immunoreactivity using mammalian antibodies for IkappaB-alpha and IkappaB kinase alpha. Furthermore, IkappaB kinase-like phosphotransferase activity, which was inhibited by the **IkappaB kinase inhibitor** sulfasalazine, was detected in brain extracts. We have evaluated the effect of sulfasalazine administration on long-term memory tested at 48 h. Amnesia was found when sulfasalazine was administered pre-training and 5 h after training but not at 0 or 24 h after training. Thus, two periods for sulfasalazine-induced amnesia were found in coincidence with the two phases of NF-kappaB activation previously described (immediately and 6 h after training). The cyclooxygenase inhibitor indomethacin did not induce amnesia when administered pre-training. Thus, the possibility that sulfasalazine induces amnesia by means of cyclooxygenase inhibition is unlikely to be tenable. In vivo sulfasalazine inhibition of basal NF-kappaB activity was found between 30 and 45 min after injection, as assessed by electrophoretic mobility shift assay. On the other hand, in vivo sulfasalazine administration 6 h after training inhibited the second phase of training-induced NF-kappaB activation, providing evidence that the sulfasalazine effect on memory is due to a direct effect of the drug on the NF-kappaB pathway. These results provide the first evidence that IkappaB kinase and NF-kappaB activation are necessary for memory formation.

L2 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 2002:190677 BIOSIS
DN PREV200200190677
TI NFkappaB activation is required for the neuroprotective effects of pigment epithelium-derived factor (PEDF) on cerebellar granule neurons.
AU Yabe, Takeshi; Wilson, Delores; Schwartz, Joan P. (1)
CS (1) NTFS, NINDS, NIH, Bldg. 36, Rm. 4A31, Bethesda, MD, 20892-4126: jps@helix.nih.gov USA
SO Journal of Biological Chemistry, (November 16, 2001) Vol. 276, No. 46, pp. 43313-43319. http://www.jbc.org/. print. ISSN: 0021-9258.
DT Article
LA English
AB Pigment epithelium-derived factor (PEDF) protects immature cerebellar granule cells (1-3 days in vitro) against induced apoptosis and mature cells (5+ days in vitro) against glutamate toxicity, but its precise mechanism is still unknown. Because the transcription factor NFkappaB blocks cell death, including neuronal apoptosis, we have investigated the ability of PEDF to exert its effects via NFkappaB activation. PEDF induced an increased phosphorylation of IkappaBalpha, decreased levels of IkappaB proteins, and translocation of p65 (RelA) to the nucleus followed by a time-dependent increase of NFkappaB-DNA binding activity in both immature

and mature neurons. The protective effects of PEDF against both induced apoptosis and glutamate toxicity were blocked by the addition of either the **IkappaB kinase inhibitor** BAY 11-7082, which inhibits the phosphorylation of IkappaB, or N-acetyl-Leu-Leu-norleucinal, which blocks proteosome degradation of IkappaB, demonstrating that NFkappaB is required for the neuroprotective effects of PEDF. Reverse transcription-polymerase chain reaction analysis revealed that up-regulation of the anti-apoptotic genes for Bcl-2, Bcl-x, and manganese superoxide dismutase was observed in PEDF-treated immature but not mature neurons. Up-regulation of nerve growth factor, brain-derived neurotrophic factor, and glial cell-derived neurotrophic factor mRNA was long-lasting in mature neurons. These results suggest that PEDF promotes neuronal survival through activation of NFkappaB, which in turn induces expression of anti-apoptotic and/or neurotrophic factor genes.

L2 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
AN 2000:58101 CAPLUS
DN 132:203533
TI Anti-inflammatory cyclopentenone prostaglandins are direct inhibitors of I.kappa.B kinase
AU Rossi, Antonio; Kapahi, Pankal; Natoli, Gioacchino; Takahashi, Takayukl; Chen, Yi; Karin, Michael; Santoro, M. Gabriella
CS Institute of Experimental Medicine, Italian National Council of Research, University of Rome Tor Vergata, Rome, 00133, Italy
SO Nature (London) (2000), 403(6765), 103-108
CODEN: NATUAS; ISSN: 0028-0836
PB Macmillan Magazines
DT Journal
LA English
AB NF-.kappa.B is a crit. activator of genes involved in inflammation and immunity. Pro-inflammatory cytokines activate the I.kappa.B kinase (IKK) complex that phosphorylates the NF-.kappa.B inhibitors, triggering their conjugation with ubiquitin and subsequent degrdn. Freed NF-.kappa.B dimers translocate to the nucleus and induce target genes, including the one for cyclo-oxygenase 2 (COX2), which catalyzes the synthesis of pro-inflammatory prostaglandins, in particular PGE. At late stages of inflammatory episodes, however, COX2 directs the synthesis of anti-inflammatory cyclopentenone prostaglandins, suggesting a role for these mols. in the resoln. of inflammation. Cyclopentenone prostaglandins have been suggested to exert anti-inflammatory activity through the activation of peroxisome proliferator-activated receptor-.gamma.. Here the authors demonstrate a novel mechanism of anti-inflammatory activity which is based on the direct inhibition and modification of the IKK.beta. subunit of IKK. As IKK.beta. is responsible for the activation of NF-.kappa.B by pro-inflammatory stimuli, the findings explain how cyclopentenone prostaglandins function and can be used to improve the utility of COX2 inhibitors.
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:511176 BIOSIS
DN PREV200000511176
TI Development of **IkappaB kinase inhibitors** as anti-inflammatory therapeutics.
AU Hottelet, Maria; Castro, Alfredo (1); Batzer, Andreas; Coggins, Michael (1); Czech, Jeorg; Dang, Luan (1); Grenier, Louis (1); Liao, Sha-Mei (1); Parent, Lana (1); Pien, Christine (1); Pink, Melissa (1); Ritzeler, Olaf; Soucy, Francois (1); Wang, Chunhua (1); Weiss, Tilo; Adams, Julian (1); Palombella, Vito (1)
CS (1) Millennium Pharmaceuticals, Cambridge, MA, 02139 USA
SO Inflammation Research, (August, 2000) Vol. 49, No. Supplement 2, pp. S91. print.
Meeting Info.: 10th National Conference of the Inflammation Research

Association Hot Springs, Virginia, USA September 24-28, 2000
ISSN: 1023-3830.
DT Conference
LA English
SL English

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